NERVE INJURIES
AND THEIR TREATMENT

PURVES STEWART & ARTHUR EVANS

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NERVE INJURIES AND THEIR TREATMENT

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NERVE INJURIES AND THEIR TREATMENT

CHAPTER I

A PERIPHERAL NERVE

STRUCTURE.

A nerve is composed of a number of nerve fibres ; each nerve fibre consists of an axis-cylinder, enwrapped in a medullary sheath which is enclosed by the neurilemma. The axis-cylinder is a prolongation of the nerve-cell body, and it extends uninterruptedly from that cell to its termination at the periphery ; it derives its existence from the nerve cell, and it degenerates as soon as its connexion with that cell is interrupted.

The **medullary sheath** is a structureless substance composed of myelin.

The **neurilemma**, or **nucleated sheath of Schwann**, is a thin, toughish membranous layer which encloses the soft substance of the medullary sheath ; on the inner surface of the neurilemma, and partly embedded in the medullary sheath, nuclei occur at regular intervals.

Midway between these nuclei interruptions occur in the medullary sheath ; these are produced by the neurilemma dipping in towards the axis-cylinder. The medullary sheath on either side of the interruption is somewhat swollen, so that the nerve fibre

presents a nodular appearance, and this region of the nerve fibre is called '**the node of Ranvier**'.

The neurilemma between two nodes of Ranvier is considered to be a single cell, the substance of which has been thinned out and wrapped round the medullated fibre, and the nucleus of this cell is that oval nucleus which lies on the inner surface of the neurilemma and is embedded in the medullary sheath.

In a peripheral nerve the nerve fibres run in small circular bundles known as **funiculi**; each funiculus has an investing sheath of connective tissue com-

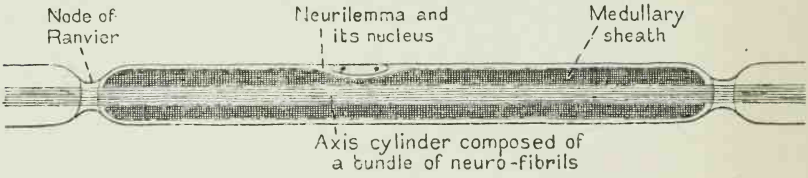


FIG. 1.—A NORMAL NERVE FIBRE.

posed of thin lamellæ of white fibrous tissue—the **perineurium**; within the sheath the nerve fibres are supported by loose connective tissue—the **endoneurium**. Large nerves consist of several funiculi; between these, and surrounding the whole nerve, is a large amount of loose areolar tissue containing numerous fat cells; in this tissue run the blood-vessels which supply the nerve structure, sensory-nerve fibres which are distributed to the nerve trunks (**nervi nervorum**), and lymphatics; to this loose connective tissue the name **epineurium** is given. See Fig. 4.

A mixed nerve trunk contains motor and sensory fibres. The axis-cylinders of the motor-nerve fibres

have their origin in the anterior cornual cells, and they pass out from the spinal cord along the anterior nerve roots.

The axis-cylinders of the sensory-nerve fibres originate in the nerve cells of intervertebral ganglia on the posterior spinal roots.

FUNCTIONS.

1. Motor fibres convey motor impulses to the muscles.

2. Sensory fibres convey sensory impressions from the periphery; these impressions are diverse, and may be represented diagrammatically thus:

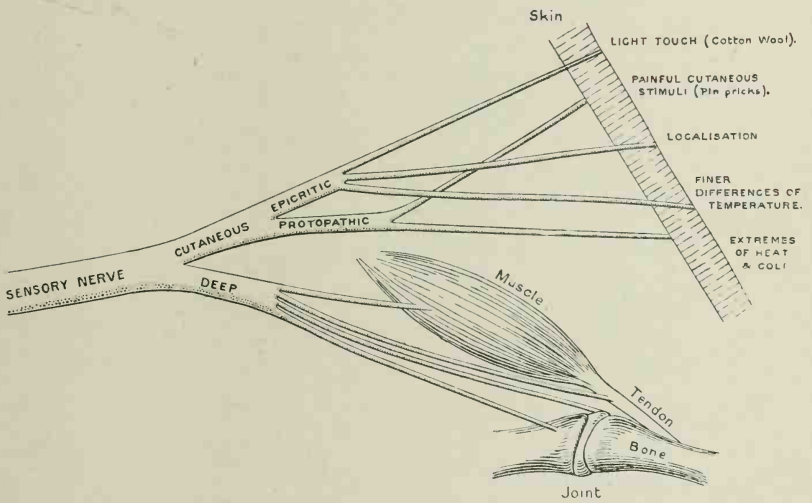


FIG. 2.—DIAGRAM OF A SENSORY NERVE.

There are three main classes of sensibility:

A. **Deep sensibility**, recognizing deep pressure, pressure-pain, joint sensation, active muscle-sensation and vibration sensation.

B. **Protopathic cutaneous sensibility**, recognizing

painful cutaneous stimuli:—pin-pricks, faradic stimulation, and extremes of heat and cold.

C. **Epicritic cutaneous sensibility,** recognizing light touches (e. g. cotton wool), cutaneous localization, and finer differences of temperature.

3. A nerve exerts a 'trophic' influence on all the structures to which it is distributed, and the nature of this influence on muscles, skin and its appendages, joints, and occasionally even the bones, is demonstrated by the changes which occur when the nerve has been interrupted. See page 8.

A DIVIDED NERVE

Alteration in Structure.

Soon after a nerve has been divided its peripheral end degenerates.

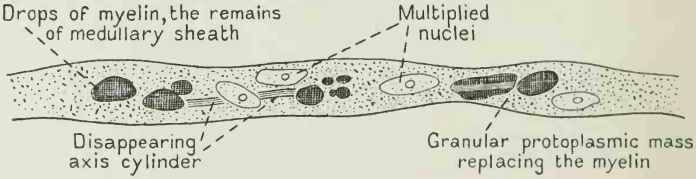


Fig. 3.—A degenerating nerve fibre.

The medullary sheath shows the first sign of degeneration, and becomes broken up into a mass of fatty globules, which are ultimately absorbed; this change in the myelin is soon followed by a similar change in the axis-cylinder, which becomes broken up and ultimately disappears. All that then remains of the nerve fibre is the neurilemma sheath, the nuclei of which have multiplied, filled by a protoplasmic mass.

The degeneration takes place simultaneously along the whole length of the nerve distal to the section, and the rapidity and completeness of the process is in no way influenced by the immediate suturing of the cut ends of the nerve.

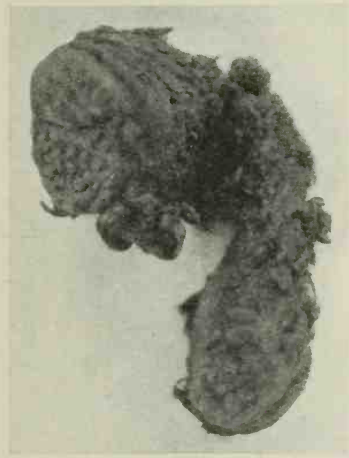


FIG. 4.—PHOTOGRAPH OF A COMPLETELY DIVIDED SCIATIC NERVE (exact size). The upper end of the photograph is the proximal end of the nerve. The two ends are connected by dense fibrous tissue. Note the slightly bulbous ending of the proximal segment, the obvious differentiation of the nerve into external and internal popliteal divisions—the upper being the external popliteal nerve. The funiculi stand out clearly in the cross section, this being particularly obvious in the internal popliteal nerve. The cut ends were directly sutured, with but slight flexion of the knee.

To the naked eye the proximal end of a cut nerve presents a bulbous end which is hard on palpation; the distal end is generally smaller than normal, and usually both ends are involved in scar tissue uniting one to the other and both to the surrounding structures.

ALTERATION IN FUNCTION.

1. **Motor.** The muscles supplied by the nerve are at once paralysed; by the seventh day they cease to respond to the faradic current, and by about the tenth day a change takes place in its response to the galvanic current (see Reaction of Degeneration, p. 21).

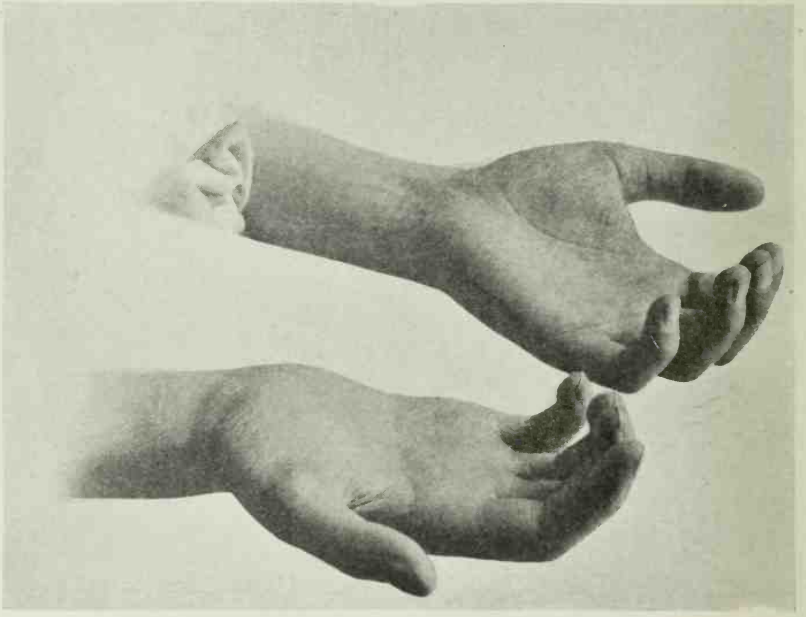


FIG. 5.—The hands of a soldier whose right upper arm had been shot through, with division of the median nerve and severe compression of the ulnar nerve. The picture shows marked wasting of the thenar and hypothenar eminences, increased growth and curvation of the nails, and wasting of the finger pads.

2. **Sensory.** The sensory area exclusively supplied by the cut nerve can no longer transmit impressions to the cerebrum; this applies not only to the cutaneous distribution, but to its distribution to muscle, tendon, bone, and joint. See Fig. 2.

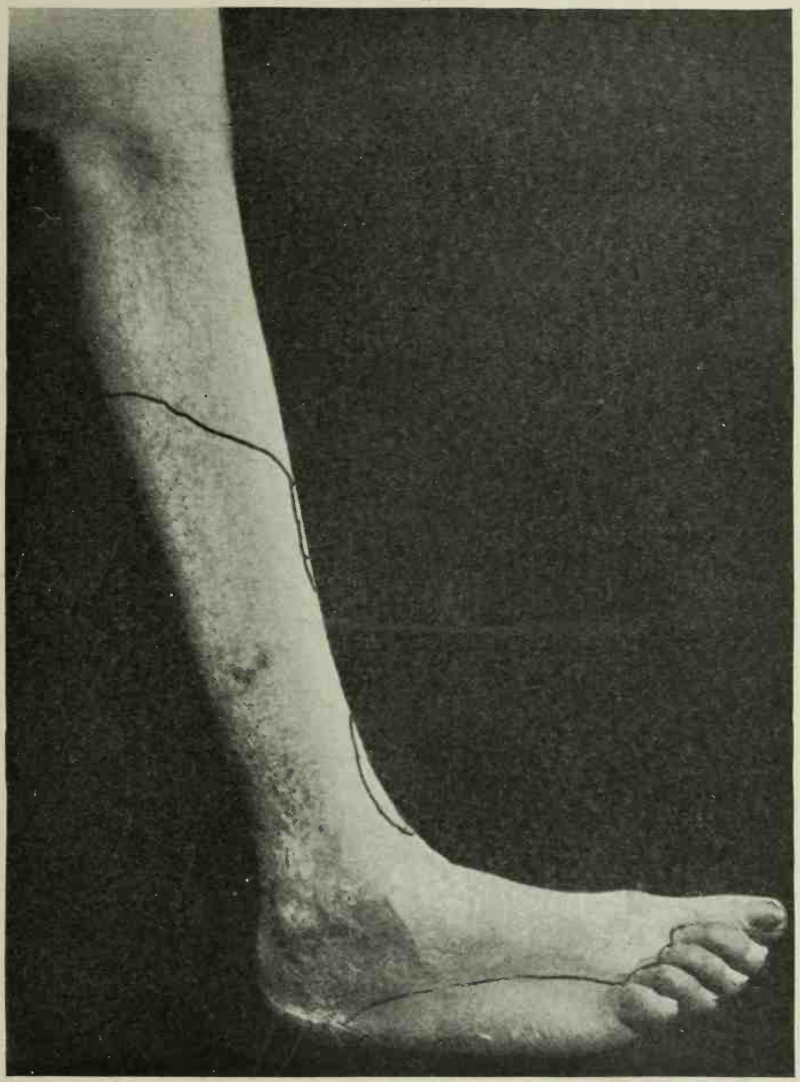


FIG. 6.—FOOT OF A SOLDIER SHOT THROUGH THE RIGHT BUTTOCK WITH INJURY TO THE EXTERNAL PERONEAL NERVE, showing delayed desquamation over the anæsthetic area—the outer surface of the leg and the dorsum of the foot.

3. **Trophic.** The muscles atrophy, unless by massage and electrical treatment their nutrition be maintained (Fig. 5); the epithelium in the insensitive area does not desquamate at the same rate as that

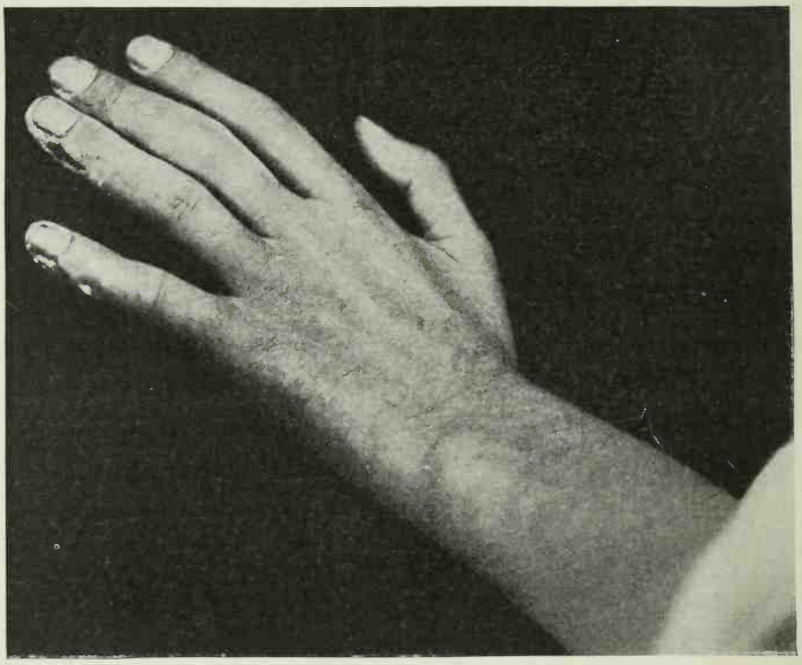


FIG. 7.—PARALYSIS OF THE ULNAR NERVE. Note the 'trophic' ulcers on the ulnar sides of the little and ring fingers. These originated in burns produced by the patient resting his hand upon a radiator, unconscious that it was hot.

over the normal skin, and scales adhere to the surface (Fig. 6).

In testing for loss of protopathic sensibility it will be noticed that pin-pricks bleed more readily in the anæsthetic area than in the adjoining territory, and leave red spots which may persist for days.

In long-standing cases the skin, especially of the fingers, becomes thin and atrophic, with a smooth, shiny surface—the so-called 'glossy skin'; the nails become longitudinally striated and markedly curved from side to side; the finger-pads are wasted, and the finger-tips taper to a point; sometimes there is a horny thickening of the epidermis in the affected area (hyperkeratosis).

Areas of skin which have lost protopathic sensibility are particularly liable to injury; it is not uncommon to find ulcers present in these situations, for being insensitive, warnings are unheeded and trauma results; and indeed, traumata too trivial to be followed by any lesion of healthy skin, under these altered conditions may result in inflammation and the destruction of tissue (Fig. 7).

RECOVERY OF A DIVIDED NERVE

Its structure.

At the end of three weeks new axis-cylinders are formed within the neurilemma sheaths by the deposition of a thin thread along one side of a spindle-celled neurilemma cell; this thread, which is beaded, grows in length until it meets the threads above and below it.

The new medullary sheath makes its appearance about the fourth week, when it too is laid down by a process of secretion along one side of a neurilemma cell. The same process takes place in the primitive end bulb and in the distal segment; in the scar

tissue intervening between the proximal and distal segments the same process takes place by the action of spider-like neuroblasts, cells which are identical with the proliferated neurilemma cells.

The same process of regeneration takes place whether the divided ends have been united or allowed to remain separated; in the latter case, however, the axis-cylinders and medullary sheaths are slower in making their appearance; they never attain to full maturity, but remain permanently in the incomplete beaded stage; if, however, secondary suture be performed subsequently, the nerve fibres soon grow to normal dimensions.

According to some observers, regeneration takes place solely by the growth of new fibres from the axis-cylinders of the central cut end; the growing axons find their way either into old neurilemma sheaths or into the interstices between them, and in this latter case fresh nucleated sheaths become formed around them.

The new fibres are at first pale, but afterwards acquire a medullary sheath, and still later a neurilemma with constrictions of Ranvier.

Its functions.

After suturing the ends of a divided nerve, the various elements in sensibility return in a definite order: sensibility to deep pressure returns first, followed by the recognition of painful cutaneous stimuli (protopathic), and lastly by the sensibility to light cutaneous impressions (epicritic).

About the time that epicritic sensation begins to reappear, motor power returns, and voluntary power is first noted in those muscles nearest the seat of the lesion, followed by its return in those more remote.

The return of motor power is followed by that of faradic excitability.

CHAPTER II

METHODS OF EXAMINATION

I. Sensory functions

A few general maxims may here be stated:

It is convenient to investigate sensory functions first, whilst the attention of the patient, and of the physician, is fresh.

We should not weary the patient by too prolonged examination, lest, as he becomes tired or impatient, his answers become inaccurate.

If no sensory changes are present, we recognize the fact in a few seconds; if changes are present they should be carefully mapped out by a skin-pencil and subsequently recorded on a graphic chart.

In the mapping out of areas of lost or altered sensation, we are largely dependent on the patient's intelligent co-operation. All sorts of ingenious apparatus have been devised for the accurate measurement of minute difference in sensibility, but the simpler our methods of examination, the more accurate are the patient's responses likely to be.

During the examination of sensory functions, the patient's eyes should be closed, so that his attention may not be diverted by watching our manipulations.

Sensation in a peripheral nerve is a complex affair, comprising three main classes of sensibility.

1. Deep Sensibility, which recognizes **deep-pressure** (e. g. by the blunt end of a pencil), which pressure, if excessive, produces a sensation of pain —**pressure-pain.** It also includes **joint-sensation** (or sense of position on passive movement), **active muscle-sensation** (kinæsthetic sense or sense of active muscular contraction), and **vibration-sensation**, produced by placing a sounding tuning-fork over the subcutaneous surface of a bone, or upon a finger-nail.

The sensory fibres, which conduct these varieties of deep sensibility, run, not in the cutaneous fibres, but in the deep afferent fibres from the muscles, tendons, and bones. So long as these deep fibres are intact, even although the skin be totally anæsthetic, the patient is able to recognize the pressure-touch and pressure-pain of a blunt pencil, the vibration of a tuning-fork, and the position of his joints on passive movement.

2. Protopathic Cutaneous Sensibility. This recognizes painful cutaneous stimuli (pin-pricks, faradic stimulation), also extremes of cold and heat. These protopathic fibres are the first to regenerate after injury to a cutaneous nerve, so that as a peripheral sensory nerve heals, protopathic sensations are the first to return.

3. Epicritic Cutaneous Sensibility, whose fibres are the slowest to recover after injury. This group includes the recognition of **light touches**

(tested by cotton-wool), of **cutaneous localization** (tested by observing the shortest distance at which the two points of a pair of blunt compasses are recognized by the patient as being separate), and the recognition of **finer differences of temperature**—not merely between hot and cold, but between warm (about 38° Centigrade) and cool (about 24° Centigrade).

Each variety of sensation should be examined and recorded separately; abnormalities of sensation should be marked on the patient's skin and then copied on to a chart.

When mapping out areas of anæsthesia, it is better to begin within the anæsthetic area and to work towards the normal skin, not in the reverse direction; as it is easier for the patient to recognize the moment when he first feels a sensation than to tell when he first loses it. On the other hand, when mapping out areas of hyperæsthesia (excessive sensibility) or of paræsthesia (altered sensibility), we should work from the normal skin towards the abnormal area, watching when the patient first notices a difference in the sensation produced.

Clinical Investigation of Sensation.

The patient's eyes being closed, we begin by testing cutaneous sensation. Touch is tested by a tuft of cotton-wool, or some similar substance which does not produce pressure. Pain is tested by pricking, or better by scratching, with a sharp needle; cold and heat by test-tubes containing ice-cold and hot

water at 50° Centigrade, or more conveniently by a couple of metal spoons just removed from jugs of cold and hot water respectively. Pressure-sense is tested by means of a pencil or other blunt object, or by a series of objects of similar size and different weights, e. g. a shilling and a sovereign. To test joint-sense, first see that the limb on the proximal side of the joint is fixed, then passively move the distal end of the limb in various directions ; finally hold it in a particular posture, e. g. flexed or extended, and then ask the patient to indicate its position. During this test we must be careful that the patient's muscles are relaxed so that he does not move the joint himself, otherwise he may gain information as to its position by means of his kinæsthetic sense. Vibration-sense is tested by means of a low-pitched tuning-fork, which is set into vibration and placed on the subcutaneous surface of a bone, or on a finger-nail. In normal individuals a characteristic vibratile thrill is felt.

Types of Anæsthesia in Nerve Injuries.

The area of anæsthesia in lesions of sensory or mixed peripheral nerves corresponds, of course, to the distribution of the affected nerve or nerves. If a purely cutaneous nerve be paralysed (e. g. the radial branch of the musculo-spiral), we have loss of cutaneous sensations, both epicritic and protopathic, whilst deep sensibility in muscles, tendons, and bones is still preserved. If a mixed nerve-trunk be paralysed (e. g. the ulnar nerve above the elbow),

muscular paralysis is superadded to anæsthesia, and the sensory loss affects not only cutaneous sensibility, but also deep sensations from the corresponding bones, joints, and tendons.

The area of cotton-wool anæsthesia in a peripheral nerve-lesion is always more extensive than the area of analgesia to pin-pricks. But as we ascend the nerve-trunk in a proximal direction, the higher the lesion, the more nearly conterminous do the tactile and pin-prick areas become, until in a posterior-root lesion, close to the spinal cord, the area of loss to pin-pricks exceeds that of anæsthesia to cotton-wool touches. Thus, as Head and Sherren put it, the nearer the lesion lies to the central nervous system, the more extensive and definite is the loss to pin-pricks; the nearer to the periphery, the greater is the loss to cotton-wool touches.

Another point of importance is, that as a mixed nerve recovers from its paralysis, sensation usually returns before motor power, and protopathic sensation returns before epicritic.

II. Clinical Investigation of Motor Functions

A. We commence by **inspection.** The following points should be noted:

1. The **posture** of the limb or affected part when at rest, e.g. drop-wrist in musculo-spiral paralysis, drop-foot in external popliteal paralysis, facial distortion in facial palsy, &c.

2. **Motor paralysis or paresis** on attempted voluntary movement. Such movement accentuates the abnormal posture of the affected part.

3. The presence of **muscular wasting** or of **compensatory hypertrophy.** After an injury it usually takes some weeks before muscular atrophy becomes evident. Compensatory hypertrophy, if it occurs, is still later in appearing.

4. Muscular **tremor** or **spasm.**

5. **Wounds, swellings,** or other **deformities** of the limb or along the course of its nerve-trunks.

By means of simple inspection we are usually able to diagnose the presence of motor paralysis. Further observations, however, are usually necessary to complete our examination.

B. **Palpation.**

1. The **bones and joints** of the affected limb should be carefully felt, tracing their outlines and gently observing their range of passive movement, so as to discover whether, after all, the deficiency of voluntary movement may be not really paralytic but perhaps due to mechanical causes, e. g. to fractures or dislocations, to adhesions or ankylosis of joints, or to inflammatory conditions (e. g. abscesses, &c.), in which any movement causes pain.

2. **Consistence** of the affected muscles, whether these are **spastic** (stiff and rigid), or **flaccid** (relaxed and soft).

3. **Voluntary movements** of the affected limb. In observing these we must be careful to test the limb

joint by joint, each single movement being separately observed. The limb above the joint under observation must be passively fixed whilst the movement is being attempted. Thus, when testing the power of flexing or extending the wrist, we must fix the forearm; when testing flexion or extension of the elbow, we must fix the humerus. Meanwhile, both by inspection and palpation, we closely watch for the occurrence of movement in the particular muscle under observation. Sometimes the tendon of insertion of a muscle may be seen or felt to contract, even although the muscle is too feeble to move the joint.

4. Suppose we find that one particular muscle or group of muscles is deficient in power, we proceed to estimate the **degree of motor deficiency.** If the weakness is slight, we recognize it by interposing some resistance, so as to load the muscles. In the case of the hand we make the patient squeeze our hand or compress a spring-dynamometer, comparing the pressure with that of the healthy side. In the case of the leg, we fix the limb with our hand and make the patient execute a movement against resistance.

If the weakness is more marked, loading of the muscles is unnecessary. The weight of the limb itself may already be too heavy a load for the muscles to lift. Thus, in weakness of the extensors of the wrist, we have drop-wrist; in paralysis of the dorsiflexors of the foot, we have drop-foot. Such postures may occasionally lead us astray unless

we are careful to eliminate the weight of the limb, since the weakened muscles may still be capable of a feeble degree of voluntary movement, although insufficient to raise the weight of the limb. To detect minimal movements, we must eliminate the effect of gravity and place the limb in such a posture that its own weight no longer comes into play. Thus, for example, in a case of drop-wrist, we place the forearm in a position midway between pronation and supination, and then see whether the patient can voluntarily contract the extensors of the wrist; or in drop-foot we hold the ankle passively dorsiflexed, and then ask the patient to lift the foot still higher; if the dorsiflexors still retain a little voluntary power, we can see their tendons momentarily stand out under the skin during this effort. Deep-seated tendons, e.g. of the hamstring muscles, may sometimes be felt to contract when they are too feeble to produce movement of a joint.

III. Electrical Examination

Faradic Reaction.

The terminals of the battery are fastened to two electrodes. The indifferent electrode, which is fastened to the positive terminal, should be a large flat one; this, after being soaked in warm saline solution, is firmly applied to some part of the body remote from that under examination. The testing electrode is smaller, usually about one inch in diameter, and is fitted with a key so arranged that

the current only passes when the key is depressed. The region to be examined is now well sponged with warm saline solution, and the testing electrode, after having first been tried on one's own thenar muscles, is applied to the muscle we wish to test, and is stroked over it until the point of maximal contraction is reached ; this is known as **' the motor point '**, and corresponds to the site of entry of the nerve into the muscle. If no contraction is obtained, gradually increase the strength of the current, and when a contraction takes place compare it with that obtained when the electrode is applied to the corresponding muscle on the opposite side.

If a muscle responds to the faradic current with a brisk contraction, the reaction is said to be normal, and we may safely assume that the lower motor neuron is normal. If no contraction can be obtained with the faradic current, we examine with the galvanic current.

Galvanic Reaction.

Normally, when a galvanic current is passing through a muscle, provided the current be not too strong, no contraction of the muscle takes place, but at the moment of making that current, or of breaking it, the muscle contracts briskly.

The contraction obtained at the making or the closing of a current when the testing electrode is the kathode, is normally stronger than when the testing electrode is the anode : this is usually expressed by the formula KCC > ACC.

Reaction of Degeneration.

When as a result of an injury or disease the lower motor neuron has degenerated, a corresponding change has taken place in the muscle fibre supplied by the nerve; it loses its fibrillar element, the element which contracts with a brisk twitch and which can be stimulated by a faradic shock; it retains its sarcoplasm, a less excitable element which contracts slowly and can still be stimulated by galvanism; but the response of the muscle differs from normal. Whereas in normal muscle the kathodal closing contraction is stronger than the anodal closing contraction, we now find either that one is as strong as the other, or that the anodal is stronger than the kathodal closing current, i. e. instead of KCC > ACC, we have KCC = ACC or ACC > KCC.

To this alteration in response to the galvanic current, and absence of response to the faradic current, the name of '**reaction of degeneration**' is given.

We sometimes meet with cases in which there is a sluggish contraction to galvanism, ACC being greater than KCC, and in which the response to faradism is diminished but not lost; this condition indicates a less severe injury to the nerve fibre, and is known as '**partial reaction of degeneration**'.

In addition to these two methods of testing muscle and nerve electrically, we have now at our disposal a third method which is almost certain to supersede the others: this is by means of the condenser apparatus.

Condenser Apparatus.

This apparatus was designed by the late Dr. Lewis Jones, and his latest pattern consists of ten condensers of capacities ranging from 0·025 of a microfarad up to 3·00 microfarads, as shown on the dial (Fig. 8).

Any one of these, by means of a commutator and a metronome, can be charged from the main and dis-

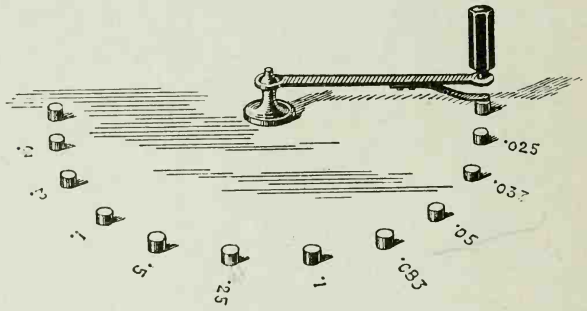


Fig. 8.—Dial of the Lewis Jones condenser apparatus. The studs are connected with condensers whose capacities range from 0·025 to 3·00 microfarads.

charged through the patient's muscle. The essential difference in these various condensers is in their wave length, i.e. the duration of their discharge through the body. The smallest condenser with a capacity of 0·025 of a microfarad takes $\frac{1}{16000}$ of a second, that with 3·00 microfarads takes $\frac{1}{130}$ of a second to pass through the body.

Healthy muscles respond to the smallest condenser, 0·025 microfarad; as a muscle degenerates it requires impulses of longer duration to make it contract; thus muscles which show partial R.D.

require condensers from 0·10 to 0·5 microfarad, and those showing complete R.D. require from 0·5 to 3 microfarads, or even more.

The advantages which the condenser method possesses over the older methods are summed up by Dr. Lewis Jones thus: firstly, it is more rapid; secondly, it is more precise; thirdly, it gives more information; and last, but not least, it is far less painful to the person tested.

CHAPTER III

INJURIES OF NERVES, THEIR METHODS OF PRODUCTION AND SYMPTOMS

THE symptoms of nerve injury may occur :

1. Immediately on receipt of trauma.
2. Subsequent to, but consequent upon, trauma.
3. Independent of any known trauma.

1. **Symptoms occurring immediately on receipt of trauma** are due to the fact that the nerve is in one of the following states :

(*a*) Divided.
(*b*) Torn.
(*c*) Contused.
(*d*) Compressed.
(*e*) Concussed.

2. **Symptoms occurring subsequent to trauma, but consequent upon it** are due to—

(*a*) Involvement of the nerve in scar tissue.
(*b*) Involvement of the nerve in callus.

3. **Symptoms occurring independent of any known trauma** are due to—

(*a*) Compression slowly developed, e. g. by new growths, aneurysms, or cervical ribs.
(*b*) Toxic conditions.

I. Symptoms occurring immediately on receipt of Trauma

(*a*) A Divided Nerve.

A nerve may be divided subcutaneously, but usually a wound of the overlying structures is present. In civil life the wound is usually occasioned by some sharp-cutting edge, as that of a knife or broken edge of glass ; in war, the division of a nerve may be due to bayonet or sword wounds, but is much more commonly produced by rifle or shrapnel bullets, pieces of cartridge-casing, pieces of shell, or by fragments of shattered bone. The nerve may be completely or partially divided.

The distinguishing feature of this group of injuries is that there is definite solution of continuity of the whole or of part of the nerve, occurring at the site of the injury and at the instant of injury.

If completely divided, there will be from that instant loss of sensation in the area supplied by the nerve, and loss of movement in the muscles supplied by it.

In about seven or eight days the muscles will no longer respond to the faradic current, and on testing by the galvanic current they will exhibit the reaction of degeneration.

If the nerve be incompletely divided, the symptoms differ greatly in individual cases. In all cases there is loss of epicritic sensibility, sometimes of both epicritic and protopathic ; in most cases there is also paralysis of all or of some of the muscles supplied by the nerve ; occasionally there is only loss of

epicritic sensibility with no motor paralysis. In some of these cases pain is the predominant feature ; the pain is in the area of distribution of the injured nerve, and begins immediately after the injury, or a few weeks later ; this area is usually tender on palpation. When such injuries are produced by rifle or shrapnel bullets or by pieces of shell these symptoms are sometimes found in a very exaggerated form ; the pain is described by the patient as ' like fire running up and down the arm ' or ' like knives sticking into the flesh '. Associated with this there may be marked atrophy of the muscles in the painful area, and sweating is often most profuse ; thus in the patient alluded to above with ' fire running up and down the arm ', the sweat literally streamed from the front of his chest and from his axilla. To this condition the name of **causalgia** (a burning pain) has been given, and in cases which have been operated upon, the nerve has usually been found incompletely divided and wrapped in scar tissue. See Fig. 27, p. 74.

These cases are sometimes described as examples of ' painful interstitial neuritis ', and the name is a fitting one.

For some days after the injury it may be impossible to state whether the division of the nerve is complete or incomplete, but Sherren has pointed out that by about the tenth day incomplete division is evidenced by what may be termed **the reaction of incomplete division** ; the muscles do not react to faradism, but to the galvanic current they respond with a weaker

current than on the normal side, the contraction is brisk compared with that seen when reaction of degeneration is present, and, as a rule, KCC remains greater than ACC.

Sherren also states that the reaction of incomplete division applies not only to incomplete anatomical division, but to all cases of **incomplete physiological division,** namely, those cases in which with no apparent loss of continuity of the nerve we have degeneration of some of the axis-cylinders below the level of the lesion, e.g. severe contusion, compression from callus, adhesions to surrounding structures, &c.

(*b*) A Torn or Lacerated Nerve.

In all other lesions of nerves the force producing the lesion is applied directly over the site of the lesion, but in this variety the force is applied more or less remotely, it is an indirect lesion. The only force which can thus act at a distance is traction, and such lesions have been well named **traction injuries.**

These injuries usually affect the brachial plexus (see p. 112). When a nerve is stretched to the breaking-point the sheath first ruptures, and this is followed, if the stretching continues, by rupture of the nerve fibres ; the rupture may be partial or complete.

The symptoms are those of a complete or incomplete division of a nerve ; usually those of complete division, for though primarily only some of the nerve fibres may be torn across, the remainder are so

damaged that degeneration ensues; or if they at first escape, are destroyed later by the organization of the blood effused in the neighbourhood.

(*c*) A Contused Nerve.

A nerve is said to be contused when it suffers alteration or loss of its functions due to trauma applied to the nerve or directly over it, when such trauma does not result in anatomical division.

The lesion in contusion varies within wide limits; it may be very severe, amounting to complete crushing of the nerve with symptoms of complete division. In less severe cases some only of the fibres are so injured, either by the trauma or later by the organization of effused blood, that they degenerate, whilst others only temporarily lose their conductivity; in these cases some muscles only are paralysed, whilst other cases escape with 'patchy' loss of sensation. In still slighter cases conductivity is not lost, or there is loss for a brief period, associated with local pain (from injury of the nervi nervorum), and numbness and tingling in the area supplied by the nerve; in these mildest cases there can be no gross lesion of the nerve.

(*d*) A Compressed Nerve.

In compression, the injuring force is slowly and steadily applied.

Pressure over a nerve gives rise to unpleasant symptoms, tingling and numbness, which symptoms cause the patient to alter the position which occa-

sions the discomfort. If, however, in spite of warnings the patient persists in the action which occasions the discomfort, e. g. the use of an improperly made crutch, or, owing to sleep or anæsthesia, he is oblivious to the warning, then these preliminary symptoms are followed by those of loss of conductivity of the nerve.

In other cases, where the pressure on the nerve is not from without but from within the limb, as in developing callus, or sclerosing fibrous tissue, or pressure over some bony deformity, degeneration of the nerve sets in with the full train of its consequences—loss of sensibility, motor paralysis, muscular atrophy, and trophic changes. It will thus be seen that the symptoms vary in severity with the duration of the compression—from the momentary discomfort experienced by pressure on the ulnar nerve as it passes behind the internal condyle, to the complete degeneration of the nerve following involvement in callus. Between these extremes we have other grades of more or less severity, with symptoms more or less pronounced, dependent on the duration of the injuring force.

As examples of injuries through compression, we have:

Momentary Pressure, e. g. on the ulnar behind the internal condyle.

Sleep Paralysis, e. g. of the musculo-spiral or ulnar nerve when the arm is hanging over the back of the chair or over the edge of a table, especially if the head be pillowed on the arm.

Crutch Paralysis, e.g. when the patient, instead of taking part of the body-weight on his hands by conveniently-placed handles, lets the whole weight fall on the axilla; here the musculo-spiral nerve is that which usually suffers, but the circumflex, ulnar, or median nerve may be simultaneously affected.

Anæsthesia Paralysis, e.g. of the external popliteal nerve from the pressure of a Clover's crutch, or of the musculo-spiral from pressure of the arm against the edge of the table.

It should be noted in fairness to the anæsthetic that it is no more to blame than is the sleep in so-called 'sleep paralysis': in both cases the paralysis is due to pressure improperly applied.

(*e*) A Concussed Nerve.

A nerve is said to be 'concussed' when its conductivity has been impaired by trauma in its neighbourhood; the nerve is not directly in the path of the foreign body inflicting the injury, and on examination no macroscopic change can be detected. It is assumed that a change has taken place in the nerve analogous to that occurring in concussion of the brain, and that the vibrations in the neighbouring tissues, caused by the rapid transit of the injuring body, have thrown the structure of the nerve into a state of 'commotion', which temporarily places its functions in abeyance.

The symptoms of paralysis follow immediately upon receipt of the injury. On careful examination it will generally be found that the paralytic symptoms are

incomplete, that the anæsthesia is transient or even absent, that the muscles are paresed rather than paralysed, and that the muscles have retained their normal electrical reactions. There is little doubt that many cases of so-called ' concussed ' nerve are in reality examples of ' functional ' paralysis.

II. Symptoms occurring subsequent to but consequent upon Trauma

These are all dependent upon compression, and the compressing force is either sclerosing fibrous tissue or bony callus. In these cases there is at the time of the trauma either no symptom suggestive of nerve injury, or slight transient symptoms consequent upon concussion or contusion—a few weeks later symptoms of compression manifest themselves and progress towards complete paralysis (p. 29).

In this group by far the larger number are due to the formation of sclerosing fibrous tissue ; this fibrous tissue results from the organization of effused blood and inflammatory exudate. It is most important to recognize this, for if, by massage, movements, and electrical treatment, we promote the rapid absorption of the extravasated blood and of the inflammatory exudate, we shall be doing much to prevent the onset of this type of paralysis—and it is an exceedingly common one.

To emphasize the importance of this, see how easy it is to manufacture a compression paralysis ! Let a patient with fracture of both bones of the forearm

be treated with splints and firmly bandaged; see that not even the fingers are free to move, and take care not to remove the splints for a month or six weeks, and if at the end of that time the hand is not paralysed it is not our fault.

Blood has been effused between and about the ends of the fractured bone, has infiltrated the muscles in that segment of the limb, has invaded the intermuscular planes and loose connective tissues, including the sheaths of the nerves and often the nerve itself; by preventing all movement of the muscles, both active and passive, and by limiting the blood-supply by tight bandaging, we have done our best to prevent the absorption of the effused products and have encouraged their conversion into fibrous tissue; and this has occurred throughout all the structures in that segment of the limb—muscles, intermuscular planes, nerve sheaths, &c.

The resulting condition is sometimes known as **ischæmic paralysis**; the muscles may react to faradism or not, there may be sensory loss or not, but there is always motor paralysis; it is all a question of the extent of the fibrosis; there is always sufficient in and about the muscles to cause their contracture and paralysis (partial or complete); there may or may not be sufficient in and about the nerve to cause sensory loss and electrical changes in the muscles.

Fig. 9 is the hand of a boy aged 11, who was seen in December 1902. Eleven weeks previously he had fractured his radius and ulna. When the splints were removed a month afterwards he found his fingers flexed and was

unable to straighten them. The hand was as shown in the photograph—fingers extended at the metacarpo-phalangeal joints and flexed at the interphalangeal joints.

The fingers were blue and cold. The fingers and the palm of the hand were anæsthetic.

On exploration the median and ulnar nerves were found adherent on their deeper surfaces; there was no massive scar formation, but for two inches the nerves were depressed

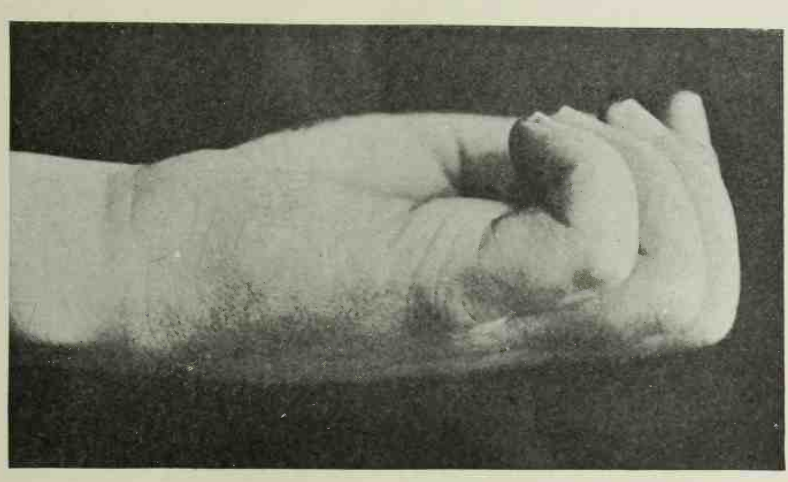


FIG. 9.—ISCHÆMIC PARALYSIS. For the treatment of a fractured radius and ulna the arm was splinted and tightly bandaged for a month. It was then looked at for the first time, when the hand presented the appearance shown in the illustration. The fingers could not be extended and were anæsthetic. There was a diffuse fibrositis at the level of the fracture—in muscles, intermuscular planes, nerve sheaths, and nerves.

below the level of the rest of their course and needed a scalpel to free them posteriorly. This was over the site of the fracture—as evidenced by the callus formation. The nerves themselves in this situation were markedly hardened—due most likely to an overgrowth of fibrous tissue outside of and between the nervous elements. Sensation and motion were speedily regained.

These cases seldom call for operative interference; if the wrist be flexed, the fingers can be straightened;

the fingers are kept extended on splints ; a few days later the metacarpo-phalangeal joints are extended and the palm and fingers are bandaged to a flat splint ; the wrist is now daily more and more extended, and kept so, on a splint, until hyper-extension is obtained. This continuous extension, interrupted only for daily massage, soon results in the cure of those cases in which the nerve symptoms are not pronounced.

III. Symptoms occurring independently of any known Trauma

These may be divided into two classes :

(*a*) **Consequent upon compression slowly produced** as by new growths, aneurysms, or a cervical rib. The commonest example of nerve paralysis produced by pressure of an aneurysm or new growth is that of the recurrent laryngeal nerve (see p. 96). For an example of nerve injury consequent on the pressure of a cervical rib see p. 132.

(*b*) The other group consists of **various forms of peripheral neuritis.** In these the condition is generally symmetrical, either both arms or both legs being affected ; in some cases all four limbs are affected.

The symptoms are both sensory and motor. The sensory symptoms consist in a diminution of epicritic sensibility in the 'stocking' and 'glove' areas ; the patient complains of tingling and of the sensation of 'pins and needles' in the same areas ; the muscles of the limbs are tender on pressure, and

this tenderness to pressure is particularly marked in the soles of the feet. The motor paralysis is most marked in the extensors of the wrists and fingers, and, when the feet are affected, the anterior tibial and peroneal muscles suffer most. This form of neuritis usually occurs as a result of some toxic absorption—alcohol, lead, arsenic, diabetes, diphtheria, or septicæmia. The neuritis of lead poisoning is not associated with sensory symptoms; there is paralysis of the extensors of the wrists and fingers, but the extensor ossis metacarpi pollicis and the supinator longus usually escape, differing in this respect from musculo-spiral paralysis.

Under this heading must also come one of the forms of **trench feet.** In these cases, as a result of deficient exercise of the leg muscles, combined with long exposure to cold and wet, a condition of peripheral neuritis develops.

Fig. 10 relates to a patient who was in the trenches from Christmas until February. The floor of the trench was sometimes frozen, but more often wet and muddy. He wore putties and boots. He possessed waterproof boots, but 'they were too much trouble, as they kept on coming off'. Three weeks before admission his heels became swollen and painful. A week later his toes became similarly swollen and tender.

On admission he had pains in the soles and in the toes, dorsal and plantar, worse at night; there was marked hyperæsthesia to pressure on the soles and over the dorsum of the toes. The feet sweated excessively. There was no cyanosis or pallor. On standing up the patient could only stand on his heels; he kept his ankles slightly dorsiflexed because of the hyperæsthesia of his soles. The patient was treated with an Esmarch's bandage, and the pain and hyperæsthesia

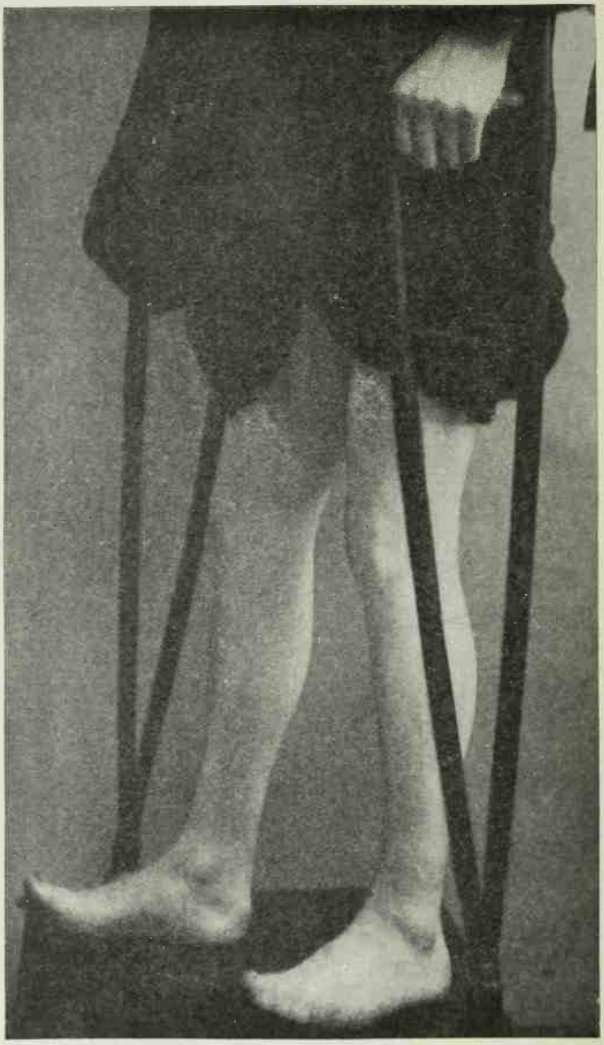


FIG. 10.—PERIPHERAL NEURITIS. One form of 'trench feet'. Notice that the patient walks on his heels; the soles of the feet are markedly hyperæsthetic; he keeps his feet dorsiflexed so as to prevent them from touching the ground.

diminished. Ten days later he could walk with crutches, resting his weight on the heels. Four days later he could walk alone, chiefly, but not entirely, on his heels.

CHAPTER IV

CONDITIONS SIMULATING A PERIPHERAL NERVE INJURY

A PATIENT is asked to make a certain definite movement, say to flex his elbow; he is unable to do so, and the question arises—Is this inability consequent on the injury of a peripheral nerve?

This inability may be due to:

1. **Some local physical condition** which makes the movement impossible, e.g. fracture, dislocation, synovitis or other inflammatory condition, adhesions, &c.; to this we have referred in describing the mode of examination of the patient (p. 17).

2. A '**functional**' cause, and not an organic nerve lesion.

3. Even if dependent on a definite organic lesion, the lesion may be in **some part of the nerve track, from cortex to peripheral distribution, other than the peripheral nerve.**

FUNCTIONAL PARALYSIS

The following points will aid in making a diagnosis:

1. In the absence of trauma the paralysis can

hardly be organic ; in the presence of trauma it may be functional or organic.

2. Muscular atrophy may occur in either, but it occurs much earlier and is more marked in organic.

3. In functional paralysis the muscles always respond to faradic stimulation and to the lowest powers of the condenser, and the reaction of degeneration is never present.

4. Organic paralysis affects definite muscles. Functional paralysis tends rather to affect movements. Paralysis of a single muscle is pathognomonic of organic disease.

5. The deep reflexes, whilst they may be exaggerated both in functional and in organic paralysis, are usually normal in functional disease.

6. Absence of deep reflexes may occur in organic, never in functional disease.

7. The combination of paralysis of the muscles supplied by a given nerve, with loss of sensation over the exact area supplied by the same nerve, can only exist with an organic affection of that nerve.

8. Functional anæsthesia never maps out the exact distribution of a peripheral nerve ; on the other hand, it is frequently bounded by a line which surrounds the limb—for instance, at the level of the shoulder, elbow, wrist, knee, or ankle ; and this line marks the abrupt termination of all sensation—protopathic, epicritic, and to deep pressure.

Fig. 11 is that of a soldier who received a gunshot wound at Ypres in May 1915. The entrance wound is on the inner side of the forearm, two inches below the inner condyle ;

he says his arm 'was slightly paralysed at the time of the accident, but under treatment with massage it soon recovered'. Three months later a piece of shell was removed, and, to quote the patient: 'The doctors had to go so deep that they cut a nerve.' His arm has been paralysed ever since.

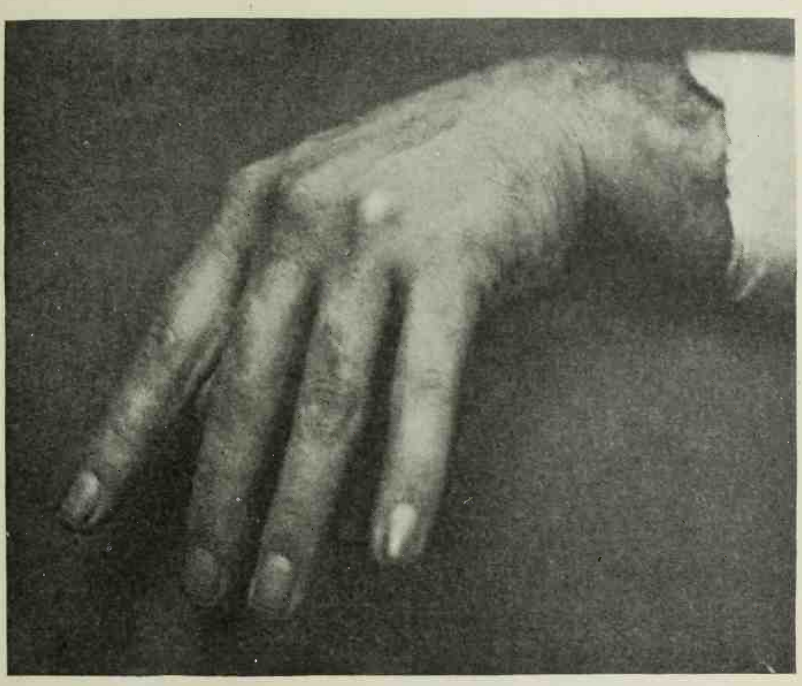


FIG. 11.—FUNCTIONAL ANÆSTHESIA AND MUSCLE-SPASM FOLLOWING A GUNSHOT WOUND OF THE UPPER PART OF FOREARM. The anæsthesia is shown in Fig. 12.

The elbow is kept flexed, the wrist flexed, the metacarpo-phalangeal joints flexed, and the inter-phalangeal joints rigidly extended (Fig. 11). There is anæsthesia of the hand and arm to just above the elbow (Fig. 12). When he is told to flex the forearm one can feel the extensors contract, and so with other suggested movements. All the muscles react to faradism. There is no muscular wasting.

A soldier was wounded by shrapnel in September 1914. The entrance was just behind the right great trochanter. Three days later the bullet was removed in Paris, through a small incision in the line of the sciatic nerve, just above the gluteal fold (Fig. 13).

January 21, 1915. He now feels the whole limb dead as far as the toes ; there is a feeling of heavy weight in the feet ; there is analgesia as high as the groin.

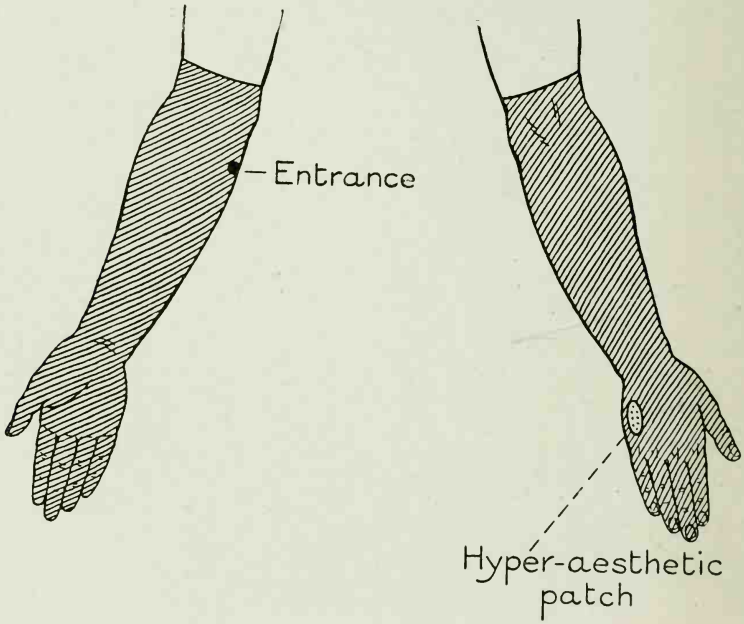


FIG. 12.—FUNCTIONAL ANÆSTHESIA. See Fig. 11.

January 31, 1915. There is anæsthesia to pin-prick and cotton-wool as high as the knee ; no muscular wasting ; right lower limb tremulous in all its movements, and feebler than the left. Gait clumsy with the right limb. Knee-jerks and ankle-jerks normal ; plantar reflexes flexor.

Fig. 14. A soldier was shot through the upper arm by a shrapnel bullet at Ypres in November 1914. The entry was in front, in the middle of the right upper arm, internal

to the biceps; the exit posterior, about the middle of the triceps. The right arm at once dropped dead.

He now complains of constant gnawing pain about one inch above the right external condyle. The elbow is habitually semi-flexed. To cotton-wool and pin-pricks there is

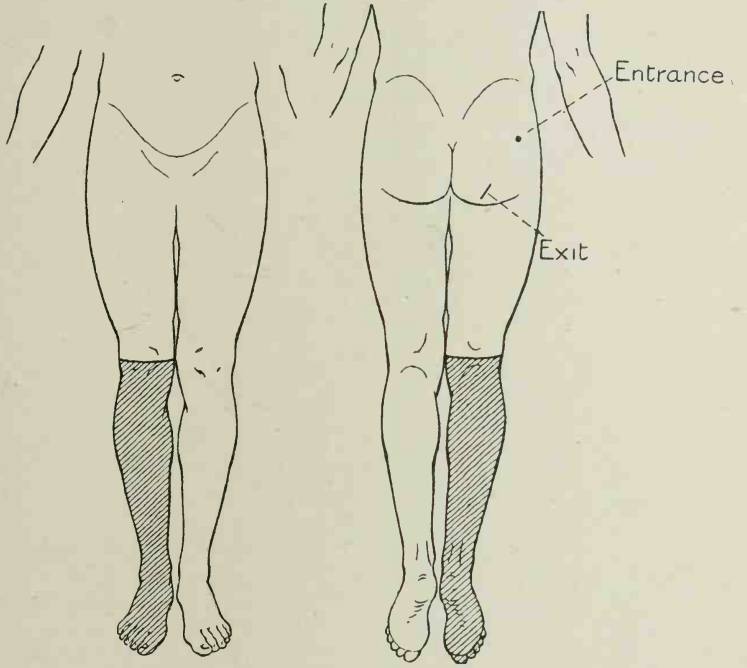


FIG. 13.—FUNCTIONAL ANÆSTHESIA FOLLOWING A SHRAPNEL WOUND. The entrance and exit are shown on the chart. When seen four months after the injury, patient said his 'leg felt dead'. There was anæsthesia to cotton-wool and pin-prick as high as the knee. A few weeks previously this had extended as high as the groin. There was no muscular wasting; all the movements of the right leg were feeble and tremulous; the knee-jerks and ankle-jerks were normal, and the plantar reflexes were flexor.

anæsthesia up to the bend of the elbow, front and back. Joint-sense is absent in fingers, wrist, and elbow. Vibration-sense is lost below the tip of the olecranon. All movements of the limb are feeble, but none impossible. Supinator jerks and triceps jerks are normal and equal. There is no muscular wasting.

Fig. 15 is that of a Belgian soldier, who during a retreat near Malines fell in a trench against a tree-trunk and bruised both shins. At the same time he was wounded by a bullet on the dorsum of the right wrist. He says he was unconscious for half an hour. He was then taken to hospital with

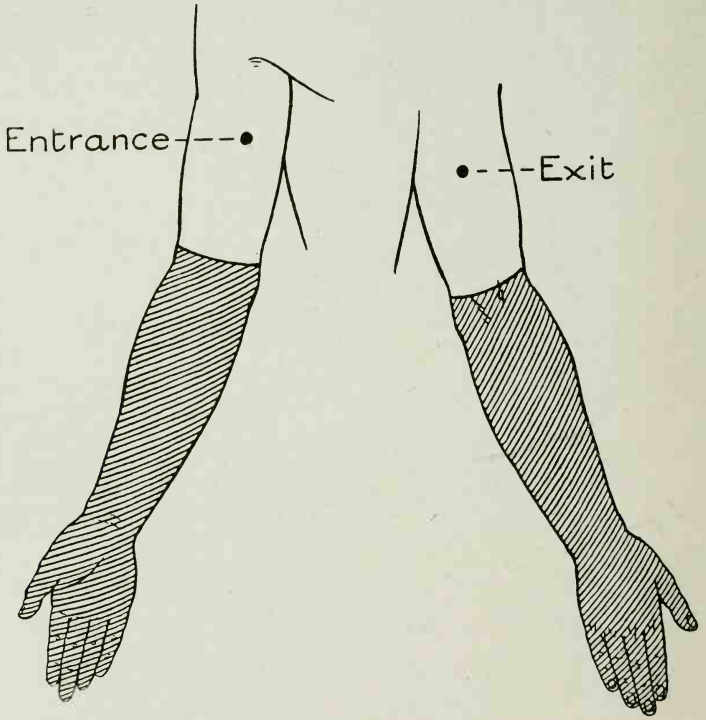


FIG. 14.—FUNCTIONAL ANÆSTHESIA FOLLOWING A SHRAPNEL BULLET WOUND THROUGH THE MIDDLE OF THE RIGHT UPPER ARM. There was anæsthesia to pin-prick and cotton-wool below the line of the elbow joint. Joint-sense was lost in fingers, wrist, and elbow; vibration-sense lost below the tip of the olecranon. All movements of the limb were feeble, but none were impossible. There was no muscular wasting.

total loss of power in the right lower limb; there was no loss of sensation. Two and a half months later, cramp developed in the limb and has since persisted.

When examined five months after the accident the right lower limb was in a condition of tonico-clonic spasm affecting all the muscles, especially the quadriceps. The hip,

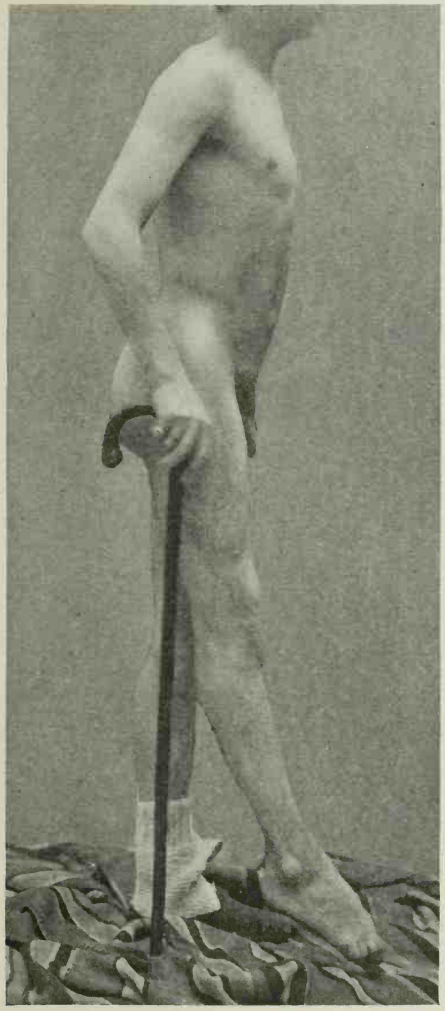


FIG. 15.—FUNCTIONAL SPASM OF RIGHT LOWER LIMB. There was no sensory loss. There was no muscular atrophy. The knee-jerks and ankle-jerks were normal.

knee, and ankle were fully extended, the toes bunched together and plantar-flexed. The limb was slightly blue from the knee downwards.

The patient resisted passive flexion of the hip, knee, or

ankle, but when considerable force was applied, all the movements could be passively carried out to the full extent, although the patient became highly emotional during the process, crying and shouting. The knee-jerks and ankle-jerks were normal, but difficult to elicit on the right side owing to the spasm. The plantar reflexes were normal. No muscular atrophy.

When the right limb was forcibly flexed, the patient could be temporarily persuaded to perform voluntary movements at all points. As soon as the limb was relaxed it at once resumed its old condition of spasmodic rigidity.

He could not walk alone, but stood on the left leg holding the right leg in a typically functional posture.

For notes of a case of functional paralysis simulating a lesion of the brachial plexus see p. 128.

Combined Organic and Functional Paralysis.

It should be remembered that a patient with an organic lesion may have a functional paralysis superadded to it. In such cases the functional element is usually more extensive in distribution than the underlying organic part and, as it were, submerges it, so that unless care be exercised in the examination, either the organic factor is overlooked and the whole case is wrongly regarded as functional, or on the other hand the organic paralysis appears to be more serious than it really is. The following is an illustrative example :

A gunner, aged 27, was hit by a shrapnel bullet at Gallipoli. The entry-wound was through the middle of the left triceps, midway between the acromion and olecranon ; the exit-wound was an inch higher up, through the middle of the biceps. The bullet-track thus crossed the line of the musculo-spiral nerve. The patient

immediately lost all power in the left upper limb, which felt to him as if it had been blown off. There was no pain at the time of injury, nor afterwards.

When examined, eight days after the injury, there was complete anæsthesia of the left upper limb to all forms of cutaneous stimulation, from the level of the acromion downwards. (See Fig. 16.) Joint-sense was absent at the

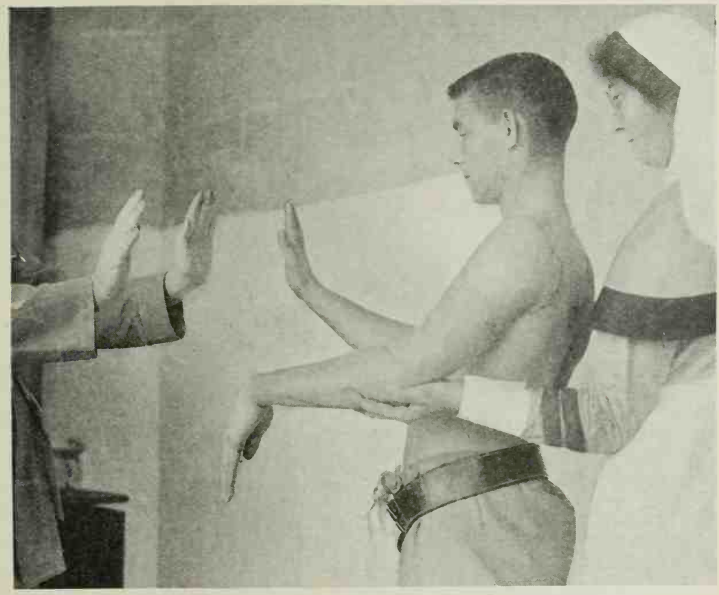


FIG. 16.—INJURY OF THE MUSCULO-SPI RALNERVE, TOGETHER WITH FUNCTIONAL ANÆSTHESIA AND PARALYSIS OF THE UPPER LIMB. The black line indicates the upper limit of the anæsthesia.

fingers and wrist, normal at the elbow and shoulder. Vibration-sense was lost in the hand, forearm, and upper arm, normal at the clavicle and scapula.

In the left upper limb the only voluntary movement that could be performed below the shoulder was feeble flexion of the fingers. No movement was possible of the thumb, wrist, or forearm. The biceps and triceps could be felt to contract feebly, but not enough to move the elbow. He could feebly abduct and adduct the shoulder.

To faradism the biceps and triceps reacted briskly,

together with all the muscles of the forearm and hand, with the exception of the supinator longus and the extensors of the wrist, fingers, and thumb.

In Fig. 16 the patient is shown during an attempt to flex the elbows and dorsiflex the wrists. On the left side he fails to accomplish this. There is wrist-drop, and the elbow is being passively flexed by the nurse. The entry-wound behind the musculo-spiral nerve is well seen. The upper limit of the anæsthesia is marked on the skin at the level of the shoulder.

Having eliminated 'physical impossibilities' and functional disease, and being left with the diagnosis of organic nerve disease, we have to decide what part of the nerve system is at fault.

The track from brain-centre to nerve-ending in muscle is clinically divided into two parts, the upper motor neurone (cortico-spinal), and the lower motor neurone (spino-muscular).

The **upper motor neurone** is that portion of the motor path which extends from the motor cortex in the cerebrum down the pyramidal tract to the nuclei of the motor cranial nerves in the crura, pons, and medulla, and further down, to the nuclei in the anterior cornua at various levels of the spinal cord.

The **lower motor neurone** is the distal part of the motor nerve track, and consists of the anterior cornual cell, its prolongation or axon in the anterior nerve root, and the peripheral motor nerve down to its ending in the muscle fibre.

The main points of difference in the paralysis occurring in these two groups is shown in the following table:

Organic Motor Paralysis

Lesion of **Upper (Cortico-Spinal) Neurone.**	Lesion of **Lower (Spino-muscular) Neurone.**
1. Diffuse muscle-groups affected, never individual muscles.	1. Individual muscles may be affected.
2. Spasticity and hypertonicity of paralysed muscles.	2. Flaccidity and atonicity of paralysed muscles.
3. May have superadded 'associated movements' on attempted voluntary movement.	3. No 'associated movements.'
4. No muscular atrophy except from disuse.	4. Early atrophy of paralysed muscles.
5. Electrical reaction normal.	5. Reaction of degeneration.
6. Deep reflexes in paralysed limbs present, and usually increased.	6. Deep reflexes of paralysed muscles diminished, and often absent.
7. If foot affected, plantar reflex is extensor in type.	7. Plantar reflex, if present, is of normal flexor type (unless flexors of toes are themselves paralysed).

Having decided that the paralysis is of the lower motor neurone, we have to determine which part of the track is affected—anterior cornual cell, anterior root, or peripheral nerve.

Anterior cornual cell and anterior root lesions

would cause pure motor paralyses unaccompanied by any sensory loss. It is difficult to imagine a trauma of the spinal cord affecting only the anterior cornual cells, and as lesions of these are usually the result of acute or chronic anterior poliomyelitis, we need not here discuss them further.

It is, however, necessary to distinguish carefully between injuries of the anterior roots and of peripheral nerves; so, too, is it necessary to distinguish between injuries of the posterior roots and of peripheral nerves.

One could not differentiate clinically between a lesion of an anterior and a posterior root and a lesion of the mixed spinal nerve formed by their fusion; the symptoms would be the same. But as soon as the nerve divides into branches to form plexuses or individual nerves we have an entirely different combination of motor and sensory phenomena which makes the diagnosis easier. In the former situation the distribution of motor and of sensory paralysis is according to root-areas, in the latter it is according to peripheral nerves. (See brachial plexus, p. 109, and cauda equina, p. 166.)

CHAPTER V

PROGNOSIS

The prognosis after primary and secondary suture will be dealt with later (p. 53). Let us first speak of prognosis in general. A patient, as the result of a wound or other trauma, presents himself with signs of a peripheral nerve paralysis; the prognosis is based on a consideration of the following factors:

1. The electrical reactions.
2. The completeness or incompleteness of the symptoms.
3. The progress of the case under treatment.
4. The condition of the wound complicating the nerve injury.

1. The electrical reactions of the muscles at the end of ten days or a fortnight.

This is of prime importance. No complete diagnosis can be made in its absence, and obviously no accurate prognosis can yet be given.

If the muscles react to faradism and respond to the lowest power of the condenser (0·025 microfarad), the prognosis is good, for we know there can be no profound alteration in the nerve, and that the condition of paralysis is either functional or consequent on disuse, and in both cases efficient treatment will speedily result in improvement.

If the muscles do not respond to faradism, and if, too, with galvanism we have polar changes—and still more, if the muscles do not respond to the higher powers of the condenser, namely, to two or three microfarads, then we know that degeneration of the nerve has taken place, and that under the most favourable circumstances three months must elapse before any return of function can manifest itself.

Between these two groups are others—namely, those in which the muscles although responding to faradism, require a stronger current than the corresponding muscles of the opposite limb. These all tend towards recovery, some more rapidly than others, and the rapidity of recovery in many cases corresponds to the ease with which they can be made to contract to condenser shocks. A muscle contracting to 0·05 microfarad is likely to regain voluntary power in a few weeks, as compared with one which will only contract to 0·25 microfarad, and which will need months for its recovery.

2. **Completeness or incompleteness of the symptoms.**

If some of the muscles supplied by a nerve still retain the power of voluntary movement, and if, too, the sensory loss be incomplete—'patchy', the prognosis is better than if the symptoms of paralysis are complete; these symptoms are sometimes the result of bruising of the nerve, with hæmorrhage into its substance.

It is in cases with a history such as this, that later

at the operation we find some thickening in the nerve in more or less of its circumference, with perhaps some limited adhesions.

Some of these cases, under energetic treatment commenced soon after the injury—such treatment being directed not only to the affected muscles but over the site of nerve injury, especially if ionization over this site be employed—show signs of improvement in three weeks or a month, as the blood becomes absorbed.

In that group of cases in which the symptoms are not profound, where we have a condition of muscle which shows paresis rather than paralysis, and in which there is no anæsthesia, or but a transitory one, we may with confidence regard them as due to 'concussion', and the prognosis is good, especially if from the sites of entrance and exit we may infer that the nerve could not have been directly injured.

3. The progress of the case under treatment.

A most important factor in determining prognosis is the response made to treatment.

Many cases under treatment will be found to improve, either quickly, as in paralysis from disuse, some cases of functional paralysis, and some concussion injuries, or more slowly, as in those cases of contusion in which the symptoms are dependent rather upon the pressure of effused blood than on actual degeneration of injured axis-cylinders.

Other cases there are in which the symptoms increase in spite of treatment, where what at first

was no more than paresis becomes definite paralysis, and in which higher and higher powers of the condenser are needed to elicit a contraction of the muscle; here it is evident we are dealing with a progressive paralysing agent. This may be either fibrosing connective tissue or callus. In these cases, apart from operation, the outlook is hopeless, and the sooner the nerve is exposed and freed from its strangulation the better.

Cases of complete paralysis, which at the end of three months, in spite of massage and electrical treatment, show no improvement, or in which examination at an earlier date reveals a nodular swelling in the course of the nerve, should be submitted to operation, as no good can reasonably be expected from further delay.

The prognosis of nerve injuries which are associated with the intense pain to which the term 'causalgia' is given, is bad, apart from operative interference, and such cases should be early submitted to operation.

4. The condition of the wound complicating the nerve injury.

The prognosis becomes much more grave when a nerve is divided or otherwise injured in a septic wound, and especially when complicated by extensive comminuted fractures of bone or wounds of joints.

The extensive injury to the nerve, the prolonged inflammatory changes in its immediate neighbour-

hood, the condition of the wound preventing any direct treatment of the nerve, and the condition of bone and joint preventing efficient treatment of muscles, tendons, and joints for a prolonged period, all combine to render the outlook as regards nerve recovery and subsequent limb utility a very grave one.

The Prognosis after Suture.

The prognosis is largely influenced by the following factors :

1. **Whether the suture was primary or secondary.**
2. **Whether the original wound was aseptic or septic.**
3. **The particular nerve under consideration.**
4. **The site of the lesion.**
5. **The treatment before and after operation.**

1. Primary versus Secondary Suture.

Assuming the operation be done under aseptic conditions, primary suture is the more favourable.

The time of return of the functions in a mixed nerve varies within fairly wide limits, and only approximate periods can be given. Speaking generally then, we may look for sensation and motion to return in the following order, and somewhere about these periods after operation.

Primary Suture.	*Commencement.*	*Completion.*
1. *Protopathic sensation*	Six weeks	Six months
2. *Epicritic sensation*	Six months	Twelve months
3. *Motor power*	Six months	Twelve months

Although epicritic sensation may be completely recovered in about twelve months, it cannot be spoken of as perfect recovery of sensibility, for it takes another twelve months before the patient acquires the power of accurate localization.

Secondary Suture.	*Commencement.*	*Completion.*
1. *Protopathic sensation*	Six to twelve weeks (may be as early as three weeks)	Six to twelve months
2. *Epicritic sensation*	Twelve months	Never
3. *Motor power*	Nine months	Very rarely perfect

In secondary suture the return of nerve functions is noticed in the same order of appearance as after primary suture. The return of protopathic sensation is sometimes observed at a much earlier date than in the case of primary suture, and this is so because by the time the secondary suture is performed partial regeneration of the axis-cylinders in the peripheral end has taken place; as a rule, however, it is later rather than earlier. Epicritic sensation takes twice the time to appear and never becomes perfect.

The dates in the above tables are those generally given, but recent experiences have shown that secondary suturing is often followed by much speedier return of sensation and motion.

Fig. 17 A is that of a case of external popliteal paralysis in which the external popliteal nerve as the result of a gunshot wound was converted into what looked and felt like a mass of fibrous tissue; this

segment was excised and end-to-end suture performed.

Five weeks later there was marked protopathic recovery. (Fig. 17 B.)

Fig. 74 relates to a soldier who as the result of a gunshot wound of the upper arm had complete paralysis of the median and ulnar nerves. At the

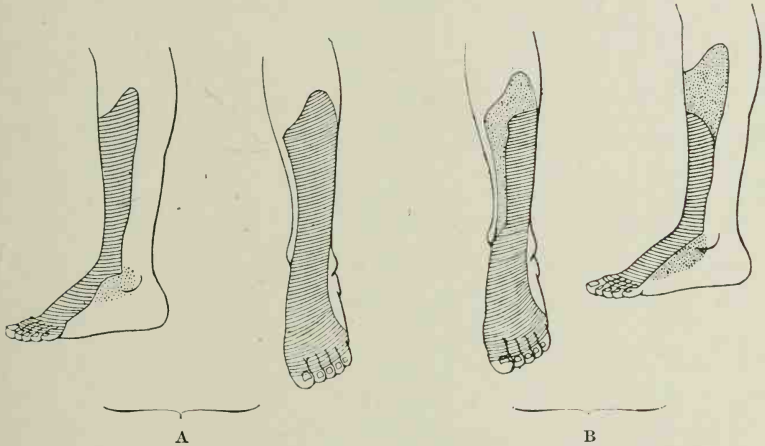


FIG. 17 A shows the loss both protopathic and epicritic in a case of complete division of the external popliteal nerve.

FIG. 17 B shows the amount of protopathic recovery five weeks after secondary suture. Over the dotted area there is only epicritic loss.

operation the ulnar nerve was found embedded in dense fibrous tissue and the median nerve almost completely torn across and the torn ends involved in massive scar-tissue. The fibrosed ends of the median nerve were excised, thus completely dividing the nerve, and secondary suture performed. Eleven weeks afterwards the patient could slightly abduct and oppose his thumb; it is worth noting that at this time there was no return of power in the flexor

carpi radialis, although, according to the rule, innervation of this muscle should have preceded that of the thumb muscles.

Bowlby states that muscular recovery is not likely to be marked if operation is delayed longer than two years, and that no case of perfect motor recovery has been reported after four years.

Sherren, reviewing twenty-one cases of secondary suture in which the interval between injury and operation was less than three years, notes that some motor recovery took place in all, but that in none was there perfect sensory recovery.

Sometimes, following secondary suture, there is no return of epicritic sensation, nor of motor power, and the only change that follows operation is return of protopathic and of deep sensibility; yet even this limited recovery is of great utility to the patient, seeing that the restoration of protopathic sensibility prevents the formation of ulcers, which sometimes originate as the result of trauma unperceived by the anæsthetic skin (e. g. a match burning to the end of its stem), and at other times as the result of injuries so trivial that they would have had no effect upon normal skin.

2. Septic wounds.

Of all the considerations affecting prognosis in nerve suture, one of the most important is the condition of the nerve with regard to its asepsis or the reverse. Thus, if in a case of primary suture for subcutaneous rupture of a nerve the wound should

suppurate—even though the suppuration speedily ceases without the nerve sutures giving way and without any sloughing of the nerve structure—recovery is much delayed, and the delay may actually double the period necessary for the restoration of the nerve functions. Again, if the original wound has suppurated, and, subsequently to its healing an operation be undertaken for suture of the nerve, even if the most favourable conditions be present, with no wide separation of the ends and no extensive matting of these to surrounding structures, recovery is much delayed. The mere fact that the wound has been the seat of suppuration exercises a markedly retarding effect upon regeneration of the nerve.

In the less favourable cases it will be found that there is wide separation of the ends, and these are involved in a mass of dense fibrous tissue which unites them to the surrounding structures.

3. The particular nerve under consideration.

The prognosis varies with different nerves. Thus, after uniting a severed musculo-spiral nerve the prognosis is good ; muscular return is almost always satisfactory, and its sensory distribution is not of prime importance, so that even if the finer shades of sensory localization fail to reappear, and the return of muscular power to the extensors of the elbow, wrist, and fingers be not perfect, still the patient will have an efficient working hand.

On the other hand, after an operation for secondary suture of the ulnar nerve, the prognosis, so far as

ultimate utility is concerned, cannot be so good; for should epicritic sensation with full return of accurate localization, and complete power of the intrinsic hand-muscles fail to appear—and this is by no means uncommon—the delicacy of touch and finer movements of the hand will be lost, and its subsequent utility much impaired.

4. The site of the lesion.

The site of the lesion, i.e. its distance from the periphery, does not much affect the return of sensation, but motor return is markedly affected; thus in wounds of the ulnar nerve at the wrist and at the elbow respectively, return of motor power to the small muscles in the hand takes twice as long in the latter as in the former case.

5. Treatment.

It cannot be too often emphasized that the results of an operation depend largely on the non-operative treatment; this applies to treatment before and after operation in the case of secondary suture, and to post-operative treatment in the case of primary suture. The almost irresistible tendency in surgery is to think, or rather to feel, that a brilliant operation has 'settled everything', or that if it has not, it ought to have!

This is practically never true in any department of surgery; it is utterly fallacious and wholly misleading in the surgery of nerves.

If the ends of a divided nerve be separated, or if

for any other reason an impermeable block bar the impulses which should pass along it, it is necessary for recovery that, some time or other, an hour or so should be spent in the operating theatre and the freshened ends brought carefully together. But in order to obtain the best possible recovery, it is equally essential that every day before that hour arrives and every day afterwards, thought should be given and time spent on keeping muscle, tendon, and joint fit and ready to respond to the impulses which one day the regenerated nerve will convey. (See p. 62.)

CHAPTER VI

TREATMENT

In considering the treatment of nerve injuries, certain facts should be borne in mind.

1. Nerve injuries are common. Expect them, and test for loss of sensation and motion.

2. The importance of early diagnosis cannot be over-estimated, for treatment should be immediate.

3. A large portion of nerve injuries tend to recover spontaneously.

4. The treatment for a divided nerve is suture, and as the prognosis of primary suture is better than that of secondary suture, primary suture is the operation of election.

5. The diagnosis of a divided nerve, immediately after an injury, is only absolute when the divided end of that nerve is seen.

6. It therefore follows that the question of immediate suture in projectile wounds will only arise when the wound is so large (either primarily from the original lesion or secondarily from the enlargement of that wound for the arrest of hæmorrhage, or suture of tendon, or treatment of a fracture) that the severed end of the nerve is exposed.

7. The great essential for the success of nerve suture is asepsis.

8. Most projectile wounds are septic.

9. In the large majority of cases many hours must

elapse before the surroundings of the patient and of the surgeon are suitable for the performance of such an operation, and by this time the wound is discharging pus ; nerve suture is now out of the question, and cannot be entertained until the wound is healed.

10. The correct treatment of a patient with a suppurating wound is the efficient treatment of the wound. Do not go fiddling with the nerve : stop the suppuration !

11. In the less extensive wounds with a small entrance and exit, such as those commonly produced by a rifle-bullet, the diagnosis of divided nerve can only be made when the characteristic signs have developed, and these are only present after ten days, and even then the signs may be dependent on conditions which do not necessitate operation ; by the time the diagnosis is established we have passed into the region of secondary suture.

12. It follows that the operation for the suture of nerves injured by projectiles is almost always secondary.

To sum up :—If the diagnosis is undoubted, i.e. if the nerve is seen to be divided, either in the initial wound or that wound enlarged, say for the purpose of checking hæmorrhage, and there is a reasonable chance of the wound being aseptic or of being made so, suture the nerve ; in all other cases wait till the diagnosis is established, and until the wound is healed.

It follows, then, from these considerations that

very few nerve injuries will be treated at once by operations directed towards the nerve; but it cannot be too often emphasized that, apart altogether from the direct treatment of the injured nerve, we must at once, and constantly afterwards, think of the other structures which are liable to serious impairment as a result of the nerve injury.

Whatever the injury to the mixed nerve, it must be remembered that changes begin to take place at once in the muscles supplied by it, in the tendons and tendon-sheaths of the muscles, in the joints which those muscles help to move, and also in the muscles antagonistic to those directly affected. Treatment must be at once directed to the prevention of these changes.

1. Muscles. The antagonistic muscles being unopposed, contract and thus shorten.

The paralysed muscles are stretched, and the longer they are allowed to remain in the stretched condition the greater is the tendency for them to remain in their elongated state. If these muscles be neglected for a sufficiently long time, the elongation becomes permanent, so that even if, by and by, the muscles regain their proper innervation and become able to contract in response to stimuli, whether voluntary or electrical, these contractions may be quite futile and result in no effective movement.

Again, apart from such merely physical effects, actual pathological changes take place in a paralysed muscle; the degenerated muscle fibres become infiltrated with fibrous tissue and fat, and become

converted into a fibro-fatty mass, so that now, instead of contractile muscle fibres, we have more or less inert, non-contractile substance.

It follows from this :

(*a*) **The paralysed muscles must not be allowed to stretch.**

(*b*) **The muscle fibres must frequently be made to contract.**

2. Tendons and Tendon-sheaths. A tendon and its sheath can only remain normal when movement of the one within the other regularly takes place.

Let a tendon remain unmoved in its sheath, and it can be taken for granted that soon the one becomes tethered to the other by numerous adhesions. These adhesions, at first fine and easily stretched, steadily become thicker, denser, and less easily stretched, until ultimately the tendon becomes fixed in its sheath.

Hence it is obvious that tendons must be frequently moved in their sheaths.

3. The Joints. The preceding remarks apply to a joint kept in one fixed position. Whatever position a joint may be in, somewhere in that joint the synovial membrane is thrown into folds, and what has been said concerning the synovial membrane in tendon-sheaths applies equally here ; adjacent folds become adherent, and the adhering folds become more strongly adherent, until by the time the patient has regained power over the muscles which should move the joint, the feebly moving muscles are unable to overcome the adhesions of the firmly fixed joint.

At the same time contraction in the lax part of the capsule of the joint is taking place with additional fixation of the joint.

It follows that paralysed joints must not be allowed to remain for a lengthy period in any one fixed position.

If once these changes which take place in paralysed muscles, immobile tendons, and fixed joints be visualized, such changes will never willingly be permitted ; paralysed muscles will be prevented from stretching and will be made to contract, whilst tendons and joints will be moved.

Non-operative and Expectant Treatment.

A limb whose nerve or nerves have been injured should be carefully wrapped up and its anæsthetic and paralysed structures protected from cold and from trauma.

The limb should be so fixed that the paralysed muscles are relaxed and prevented from being stretched by their unopposed antagonists.

Thus in wrist-drop the hand should be kept dorsiflexed, and in injuries of the great sciatic or external popliteal nerve with drop-foot, the foot should be fixed at a right angle to prevent stretching of the paralysed anterior tibial muscles.

Daily the limb should be removed from the splint and massaged, the massage being applied not only to the affected muscles, but also in the region of the nerve injury.

Electrical treatment, too, is a valuable accessory.

With regard to the form of electricity to be employed, faradic shocks should be used if the muscles react to faradism; if they do not, then the galvanic current should be employed. The current used should be just strong enough to produce contraction in the affected muscles, not enough to cause pain. Better than these two is the use of condenser-shocks applied rhythmically by means of a metronome; these are painless, and in their highest powers (2·5 and 3·0 microfarads) cause contraction of muscles which fail to respond even to galvanism.

Electrical treatment over the site of nerve injury is also a valuable aid to recovery; this is best employed in the form of ionic medication.

Under such treatment, in cases in which the nerve has escaped anatomical division and its fibres are merely concussed, signs of recovery soon manifest themselves, in some cases in a few days, in others in a few weeks; these are the cases in which the paralytic symptoms were incomplete, the anæsthesia transient or even absent, and the muscles paresed rather than paralysed, and in which the muscles had retained their normal electrical reactions.

Somewhat more severe are the cases of contusion without loss of anatomical continuity, where blood has been extravasated around and within the nerve-sheath. In these cases a certain degree of muscle wasting may occur, but some electrical reaction to faradism can usually be obtained in one or more of the paralysed muscles, although stronger shocks are necessary than in the corresponding muscles of

the uninjured limb; in such cases improvement often sets in under treatment within three or four weeks as the blood becomes absorbed, sensation clearing up before motor power begins to return.

In other cases, namely, those in which the nerve has been partially cut across, the remainder of the fibres either escaping altogether or being temporally concussed, or contused with accompanying blood infiltration, part of the paralytic phenomena will clear up under treatment, leaving a residuum of paralysis which may be dealt with surgically.

Other cases will show from the beginning complete paralysis of motion and sensation, with reaction of degeneration in ten days, and no sign of improvement at the end of three months in spite of assiduous treatment. These are usually cases of complete section of the nerve, and frequently, examination over the site of injury will reveal an enlargement on the stump of the proximal end. There is another and larger group of cases resistant to treatment, comprising those nerves which are compressed by fibrous tissue, by newly formed callus, or by bony projections; in the case of sclerosing fibrous tissue and callus formation, the symptoms which may not have been very pronounced at first, later become complete.

A final group, not a large one, also resistant to treatment, consists of those cases which from the time of injury or soon afterwards are characterized by the intense pains and trophic changes to which we have alluded under the name of causalgia (p. 26).

These last three groups demand operation; the last group as soon as the symptoms are diagnosed as typical of causalgia, for these we know are always dependent upon incomplete division of a nerve trunk; the compression group as soon as the progression of the paralytic symptoms reveals the nature of the lesion; and the other group when, in spite of active treatment, the three months' probation has passed with no sign of recovery manifesting itself.

Operations on Nerves.

In all operations on nerves asepsis is imperative. Fine catgut sutures should be used, and the finest round-bodied needles which will carry the catgut.

The knives used should be the sharpest possible, and the nerves should be treated with the greatest possible tenderness.

The nerve should never be held in a dissecting forceps; the sheath gives all the necessary 'hold' one can require.

If the nerve needs to be lifted up, pass a strip of gauze under it and lift it up by means of this. (See Fig. 29.)

In performing end-to-end suture the ends should be brought together without tension; one stitch should be passed through the whole thickness of the nerve-ends, and sheath should be brought to sheath by two, three, or more quilted sutures, the number increasing with the size of the nerves (Fig. 18).

The line of suture and the adjoining portion of the nerves should invariably be protected, either by

enclosing them in a piece of excised vein or by an investment of fascia or fat, or by a sheet of sterile cargile membrane carefully wrapped round. This latter can be strongly recommended.

The greatest care should be taken to stop all bleeding, and the wound should be 'dry' before it is sewn up.

Should oozing persist in spite of all efforts to stop it, insert a drainage-tube for twenty-four hours.

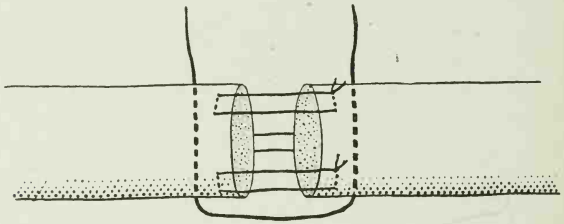


FIG. 18.—DIRECT NERVE SUTURE. One catgut suture passes through the whole thickness of the nerves, and quilted sutures bring sheath to sheath.

1. The nerve may be wholly divided and the ends retracted.

The bulbous end should be removed; the distal end in the absence of a bulbous enlargement should be 'freshened', and the new surfaces brought in contact by fine catgut sutures.

2. The cut ends of the nerve may be so widely separated that it is not possible to bring them together.

The following methods may be employed:

(*a*) *Flexion of the joint over which the nerve passes.* This is particularly useful in operations on the great sciatic, popliteal, and median nerves.

It need hardly be said that the amount of flexion must not be such as to seriously impair the utility of the limb.

Fig. 19 is that of a gunshot wound through the lower end of the right femur. The bullet, having perforated the femur, has divided the sciatic nerve ; the ends are separated by a gap of $3\frac{1}{2}$ centimetres. It will be seen that the proximal end of the nerve terminates in a large bulb, and a smaller bulb is

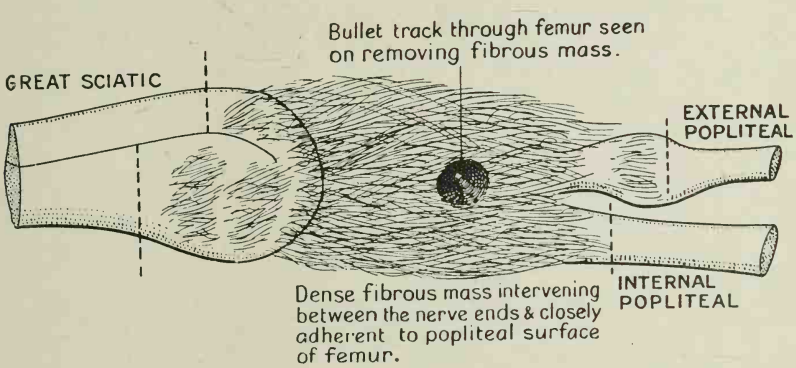


FIG. 19.—RIFLE-BULLET WOUND OF THE GREAT SCIATIC NERVE. The dotted lines show the portions excised before suturing the ends together. Fig. 89 shows the small amount of flexion of the knee which permitted the direct suture of the ends.

situated in the external popliteal nerve very near to its severed end. There is no bulbous enlargement of the internal popliteal nerve.

The fibrosed ends of the nerves were removed together with the mass of dense fibrous tissue uniting each to the other and both to the posterior surface of the femur.

In spite of the wide separation (7 centimetres after the ends had been freshened), the ends were easily

brought together after stretching the proximal end and slightly flexing the knee.

Fig. 89 shows the leg five weeks after the operation, and the small amount of flexion of the knee.

(*b*) *Altering the position of the nerve so as to shorten its course.*

This is especially applicable to the ulnar nerve.

Fig. 20 represents the two ends of the ulnar nerve following a gunshot wound at the bend of the elbow.

The proximal portion of the nerve ended in a slight enlargement just above the internal condyle, and

FIG. 20.—The two ends of the ulnar nerve following a gunshot wound at the bend of the elbow.

for 5 centimetres above this, the nerve was distinctly thickened and was harder than normal, due apparently to hæmorrhage into the nerve and subsequent fibrosis.

Below this, and filling up the ulnar fossa behind the internal condyle, was nothing but fibrous tissue, and the distal end of the nerve was found on dissecting in the interspace between the two heads of the flexor carpi ulnaris. The ends were separated by 4 centimetres.

The gunshot wound had resulted in a partially flexed elbow joint. The ends of the nerve were sutured directly together on bringing them in front of the internal condyle (Fig. 21).

(*c*) *Transplantation.*

A sufficiently long segment of one of the patient's sensory nerves is excised and interposed between the separated ends of the paralysed nerve.

The musculo-spiral nerve lends itself readily to this procedure, for if the nerve be followed down to its division into radial and posterior interosseous, a segment may be taken out of the radial nerve sufficiently long to bridge any gap likely to exist between the ends of the divided nerve, with little or no anæsthesia resulting in the radial area (Fig. 22).

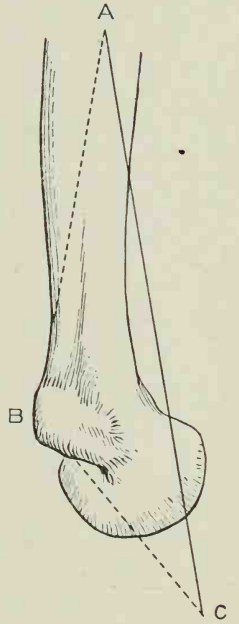


FIG. 21.—The inner aspect of the lower end of the humerus, showing the normal route of the ulnar nerve A B C, and the new shorter route A C, when the ends of the nerve are sutured in front of the internal condyle.

(*d*) *Bridging.*

This method is only adopted if for any reason the two preceding methods are impracticable.

The ends of the nerves are brought as near as possible together by catgut sutures, and then the ends of the nerves and the intervening sutures are protected by an investing tube (Fig. 23).

This investing tube is preferably a segment of one of the patient's cutaneous veins; failing this, the following have been used: decalcified bone tubes, metal tubes, cargile membrane, and better

than these, formalinized arteries which have been preserved in alcohol.

(*c*) *Nerve anastomosis.*

The peripheral end of the paralysed nerve is united to the cut surface of a neighbouring healthy nerve.

This healthy nerve is either partially or wholly divided.

Fig. 24 represents a gunshot wound of the fifth cervical root. There was no available proximal end

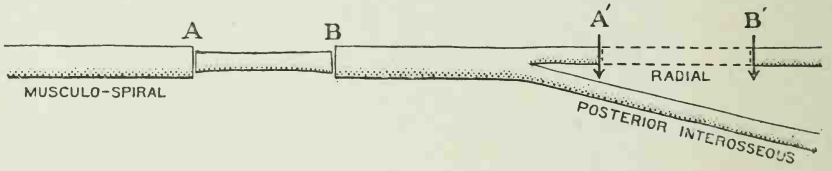


Fig. 22.—A portion of the radial nerve transplanted between the separated ends of the musculo-spiral nerve.

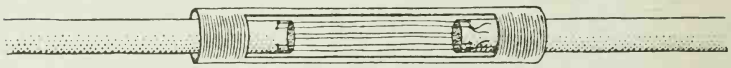


Fig. 23.—Diagram of a nerve-bridging operation. The ends are brought as near as possible to each other by catgut sutures, and the whole is invested in a segment of a cutaneous vein.

to which the distal portion could be united, so it was anastomosed to the adjoining sixth cervical root.

Care should be taken that the cut surface of the paralysed nerve is united to the proximal face of the incision (Fig. 25).

To suture it to the distal face of the incision, as in Fig. 26, would be obviously wrong, for here we are interposing the sheath of the paralysed nerve between its own axis-cylinders and those of the cut central

end, and helping to frustrate the sole object of the operation.

Another example of nerve anastomosis is the operation for facial paralysis—in this the distal end

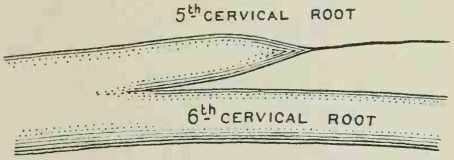


FIG. 24.—A gunshot wound of the fifth cervical root—reducing the proximal end to a fine fibrous strand.

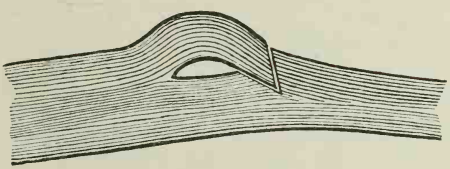


FIG. 25.—Correct method of suturing the freshened end of the fifth root to the proximal face of the incision in the sixth root.

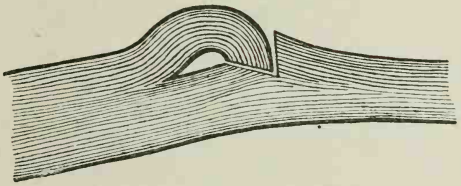


FIG. 26.—The incorrect method of uniting the nerves in lateral anastomosis.

of the facial nerve is sutured to the central end of the hypoglossal nerve.

3. A part only of the nerve may be shot away; this part may be any proportion of the whole nerve.

Fig. 27 A is that of the fifth cervical nerve, showing the upper edge shot away; the upper boundary of

the lesion was somewhat bulbous; the site of the lesion was closely adherent to the fibrosed scalenus anticus.

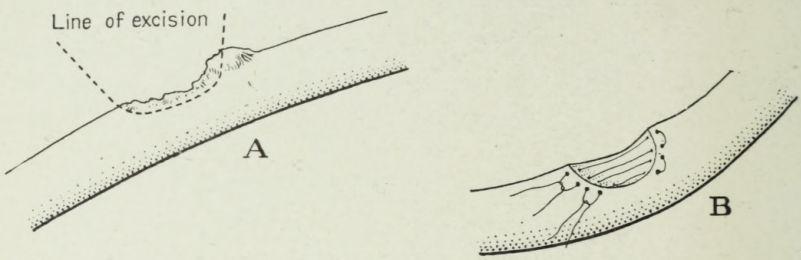


FIG. 27.—A, The fifth cervical nerve, its upper edge shot away. B, The indurated edge excised, and the proximal half being sutured to the distal.

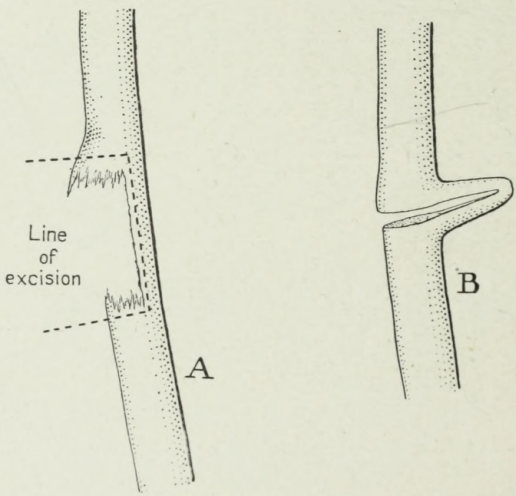


FIG. 28.—A, The median nerve, which has lost the greater part of one segment; a narrow strand maintains the continuity. B, The median nerve; its torn and fibrosed edges have been removed, and the raw surfaces are ready for direct suture.

The treatment consisted in freeing the nerve from its adhesions, excising the indurated edge, and suturing the proximal half of the freshened surface to the distal (Fig. 27 B).

Fig. 28 A is that of a median nerve in which a narrow strand alone maintains the continuity.

Treatment consists in paring the torn and fibrosed edges and suturing these together (Fig. 28 B).

4. The nerve for a greater or shorter distance may be embedded in more or less dense fibrous tissue.

The treatment consists in making a clean longitudinal incision through the fibrous tissue down to the nerve and shelling it out of its bed.

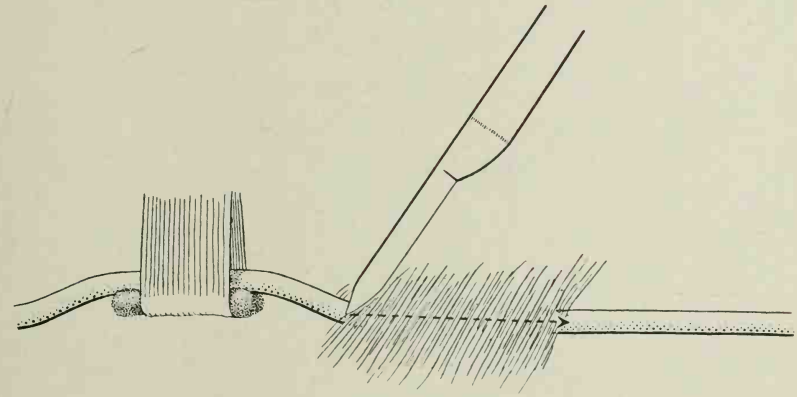


Fig. 29.—The ulnar nerve for a distance of four centimetres embedded in fibrous tissue. A piece of ribbon gauze is passed under the normal nerve; by means of this the nerve is lifted up, and a sharp knife cuts through the fibrous investment.

This is best done by passing a strip of ribbon gauze under the nerve proximal or distal to the adherent segment; by this the nerve is lifted up towards the operator, and a sharp knife carefully used, easily cuts through the fibrous investment without injuring the nerve, and at the same time the nerve is lifted out of its fibrous bed. The nerve

is less likely to be sharply kinked and injured by the ribbon gauze if a small pad of gauze be placed between the two (Fig. 29).

The nerve is now wrapped up in cargile membrane.

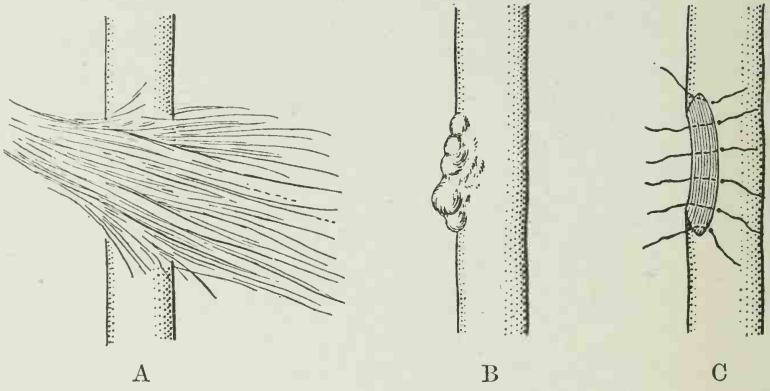


FIG. 30.—INNER CORD OF BRACHIAL PLEXUS. A, Scar tissue radiating from coracoid region to under surface of pectoralis minor. This, incised longitudinally down the middle of the nerve, easily peeled off. B, On the deep surface, the nerve was closely adherent to a mass of fibrous tissue—in which was the obliterated and fibrosed axillary artery. C, The slightly nodular, thickened, raw area was excised, and the wound closed with fine catgut sutures.

5. **Sometimes on attempting to lift a nerve out of its fibrous bed we come upon a situation where this is impossible, and here the nerve is intimately adherent at one part of its circumference to a dense mass,** the track of the bullet.

The nerve should be dissected off this fibrous bed, and the raw surface closed with fine catgut sutures (Figs. 30 A, B, C).

6. **The nerve may be found closely adherent to some adjacent structure from which it can be separated intact, but in the region of the adhesion marked thickening can be felt in the nerve.**

Fig. 31 is that of the great sciatic nerve in the lower third of the thigh. The external popliteal nerve was closely adherent on its outer and deeper edge to the posterior surface of the femur (31 A).

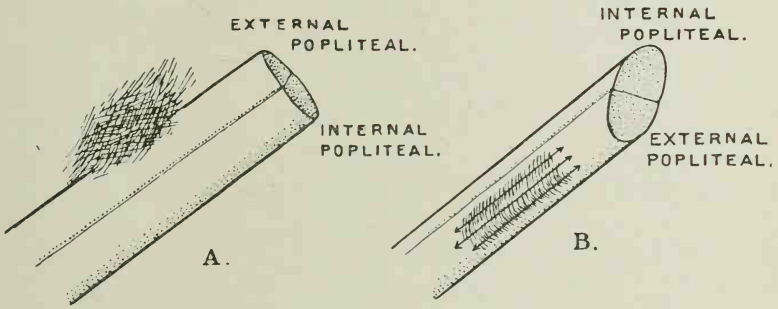


FIG. 31.—SCIATIC NERVE. A, Outer edge and adjoining portion of deep surface adherent to periosteum, with marked thickening in that portion of the external popliteal. B, The nerve freed from adhesions. Three longitudinal incisions are made through the indurated area, extending beyond its limits, and deep enough to go through its substance.

This was stripped off, and through the indurated area longitudinal incisions were made (31 B) and the nerve wrapped in cargile membrane.

7. **It is not uncommon to find in one wound diverse lesions of several nerves.**

This is particularly common in wounds of the brachial plexus, and of the upper half of the arm. The following are two examples :

Fig. 32 represents a gunshot hole in the humerus filled with fibrous tissue, in which are embedded the ulnar and internal cutaneous nerves, the separated ends of the divided median nerve, and the fibrosed termination of the brachial artery.

The treatment consisted in releasing these from their fibrous bed, freshening the ends of the median nerve and direct suture, and protecting the ulnar and internal cutaneous nerves by wrapping them in cargile membrane.

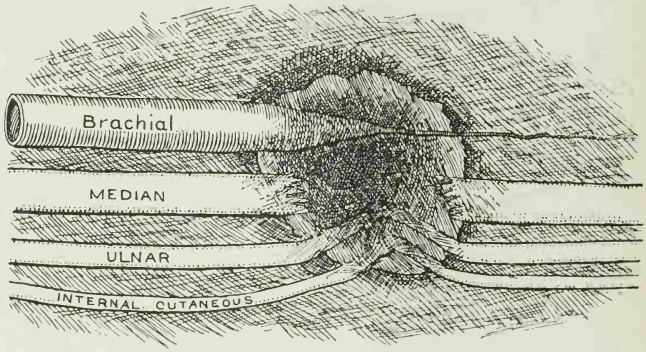


Fig. 32.—A gunshot wound of the humerus, with obliteration of the brachial artery, division of the median nerve, and compression of the ulnar and internal cutaneous nerves.

Note the condition of the brachial artery rapidly tapering to a fibrous extremity. When first exposed the altered artery and the structures in its neighbourhood, including the nerves, formed one elongated fused mass, from which the various constituents had to be dissected free. It is by no means rare to find this condition of fibrosed obliterated artery; and, when associated with nerve injuries, it is usually the axillary or brachial artery which is thus affected, and for the obvious reason

that these vessels are intimately related to main nerve trunks throughout the whole of their course.

Fig. 33 represents a composite injury of the brachial plexus. The seventh cervical nerve was divided on its inner side; the other nerves were

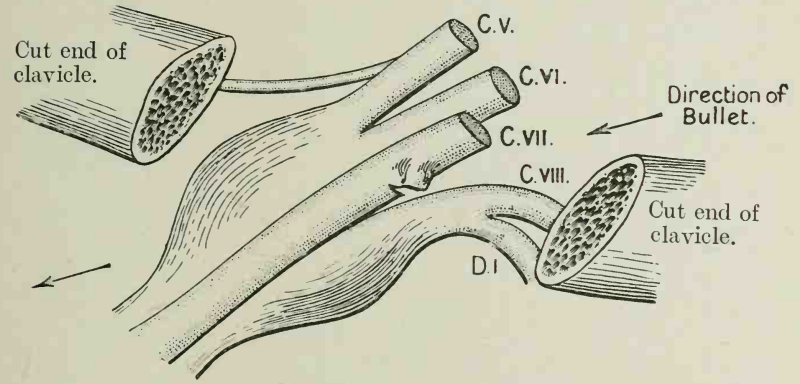


FIG. 33.—A gunshot wound of the brachial plexus.

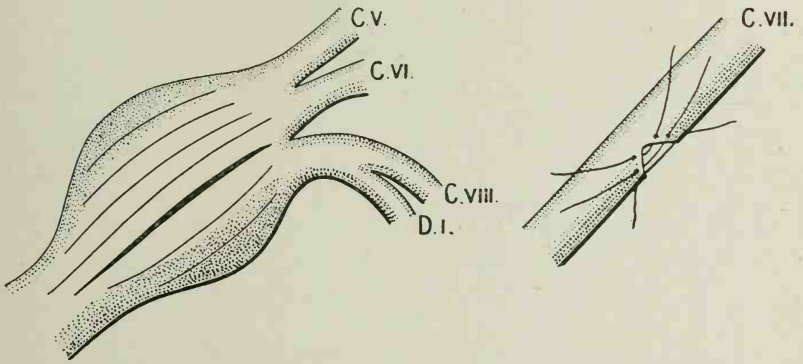


FIG. 34.—Treatment of the composite lesion shown in Fig. 33. The better to illustrate the treatment, the seventh cervical nerve is figured as separate from the rest of the plexus.

fused into a markedly fibrosed mass. All these structures were connected by adhesions to the posterior surface of the clavicle.

The treatment consisted in freeing the mass from the surrounding adhesions, then in excising the

edges of the lesion in the seventh nerve, and suturing the proximal to the distal surface of the lesion. This was followed by multiple longitudinal incisions into the substance of the fibrosed mass (Fig. 34).

8. **A piece of metal or other foreign body may be lodged in the nerve.**

Fig. 35 is that of a small piece of shell partially dividing the internal cutaneous nerve and lodging

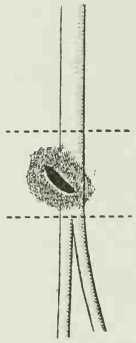


FIG. 35.—INTERNAL CUTANEOUS NERVE. Partially divided by a piece of shell, which is embedded in a mass of fibrous tissue—the undivided portion of the nerve is also fibrosed.

in it, embedded in a mass of fibrous tissue; the undivided portion of the nerve is also fibrous.

The treatment in this case is to cut out the piece of nerve involved and suture the divided ends.

9. **Lastly, a segment of a nerve may, to touch and sight, seem profoundly altered**; it feels like a mass of fibrous tissue; on section it cuts like one. The continuity is unimpaired, but there is total paralysis in the region and structures supplied (Fig. 36).

Around the treatment of this type of lesion much controversy has raged and still is raging.

The question for solution is this: Should the thickened area be cut out and the apparently normal ends sutured together, or is it better merely to free the segment from its surrounding adhesions and prevent subsequent adhesion by wrapping in fat, fascia, or cargile membrane ?

In other words, is the thickened area a mass of fibrous tissue which will interfere with the regenera-

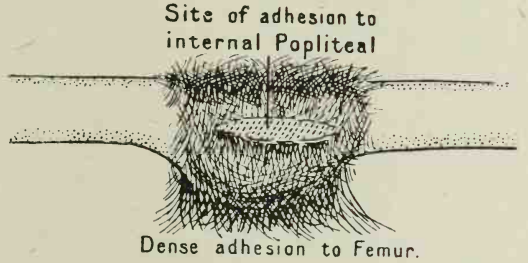


FIG. 36.—A GUNSHOT WOUND OF THE EXTERNAL POPLITEAL NERVE. The injured segment looks, feels, and cuts like a mass of fibrous tissue.

tion of the nerve, or are there still nerve-fibres in it which are in process of linking up the two ends, and is the fibrous tissue serving a useful purpose by uniting the ends ?

Assuming that the mass were nothing but fibrous tissue, it would obviously be better to substitute for that massive fibrous barrier the thin line of junction caused by carefully suturing the ends after excision of the fibrous area. But it must be borne in mind that sight and touch are not capable of excluding the presence of nerve

structures. Three more exact methods have been adopted.

1. Electric stimulation of the nerve above the fibrous zone, and noting the presence or absence of muscular response.

2. Excision of a small piece of the fibrous zone and immediate microscopic examination for nerve structure, teasing the fragment in a 5 per cent. solution of osmic acid.

3. Injection into the proximal end of the scar area of 1 c.c. of 1% methylene blue, and observing the infiltration of the coloured solution through the suspected mass into the nerve distal to the lesion (scar tissue does not permit the passage of methylene blue: nerve structures do).

By the employment of these methods it is found that in the large majority of cases healthy nerve-fibres can be demonstrated in areas which to the naked eye seem inert and useless. According to some workers, therefore, the treatment of this class of case would consist in liberating the fibrosed nerve from its surroundings, and protecting with fascia or cargile membrane.

Notwithstanding what has just been written, a surgeon meeting with such a fibrosed segment is quite justified in cutting it out, provided it is possible to perform direct suture of the cut ends. Having performed both types of operation and watched the subsequent progress, we would go further than this, and say that if one has waited for three months with no return of nerve-function,

and finds at the operation such a fibrosed segment, it should be cut out if it is possible to perform direct suture of the cut ends. If the altered segment be too extensive to permit direct suture of the cut ends after excision, then be content to free the nerve, protect it, and wait another three months. If after this further waiting period there should still be no return of function, excise the affected portion, and complete the operation by transplantation, anastomosis, or by bridging.

CHAPTER VII

LESIONS OF INDIVIDUAL NERVES

THESE will be considered under the following headings:

1. Cranial nerves.
2. Cervical sympathetic.
3. Cervical plexus.
4. Brachial plexus and nerves of the upper limb.
5. Thoracic nerves.
6. Cauda equina and nerves of the lower limb.

The individual nerves differ greatly as regards the frequency with which they sustain injury, as shown in the following table of nerve injuries seen by us since the outbreak of the war.

STATISTICS OF 316 CASES OF NERVE INJURIES

CRANIAL NERVES:

Optic		0	42
Ocular (3rd, 4th, and 6th)		3	
Trigeminal		5	
Facial		12	
Auditory		0	
Vagus (recurrent laryngeal branch)		1	
Spinal accessory		5	
Hypoglossal		3	
Multiple	2nd and 5th	1	
	3rd and 7th	1	
	5th and 7th	2	
	5th and ocular nerves	3	
	5th, 7th, and ocular nerves	2	
	7th and 10th	1	
	7th and 12th	1	
	5th and 12th	2	

Cervical Plexus	1	1
Upper Limb:		
Brachial plexus	61	
Circumflex	0	
Median	20	
Ulnar	34	
Musculo-spiral	36	
Posterior interosseous	1	
Radial	1	
Wrisberg	1	
Multiple		181
Median and ulnar	14	
Median and musculo-spiral	5	
Ulnar and musculo-spiral	2	
Median and radial	1	
Musculo-spiral, ulnar, and musculo-cutaneous	1	
Median, ulnar, and internal cutaneous	1	
Median and internal cutaneous	1	
Ulnar and internal cutaneous	2	
Trunk:		
Thoracic nerves	4	4
Lower Limb:		
Cauda equina	11	
Lumbro-sacral plexus	7	
Sciatic — Trunk	21	
Sciatic — Internal popliteal	1	
Sciatic — External popliteal	26	
Posterior Tibial	1	
Obturator	0	
Anterior crural	5	
Internal saphenous	5	88
Musculo-cutaneous	2	
Small sciatic	3	
Multiple		
Sciatic and anterior crural	4	
Great and small sciatic	1	
Obturator and anterior crural	1	
		316

Cranial-Nerve Paralyses

Notwithstanding the very great frequency of wounds of the head, cases of paralysis of cranial nerves are relatively uncommon. The reason for this is obvious: wounds which traverse the base of the skull are so often fatal that the patient rarely survives to show cranial-nerve palsies.

Most cranial-nerve paralyses are seen in extra-cranial wounds, where the nerves are implicated after their exit from the cranial base, e.g. in wounds of the orbit, face, mastoid region, and upper part of the neck.

A few cases are due to fractures running down into the cranial floor and damaging the nerves at their foramina of exit; other cases, again, are due to intra-cranial hæmorrhages, e.g. at the front of the pons or medulla.

First, or Olfactory Nerves.

On each side the olfactory nerves arise from the under-surface of the olfactory bulb, as it lies upon the cribriform plate of the ethmoid bone. The nerves pass through the small apertures in the cribriform plate and are distributed to the upper part of the nasal septum, and of the outer wall.

These nerves may be injured in fractures of the anterior fossa of the skull.

The sense of smell is tested by holding aromatic substances such as oil of cloves, peppermint, or asafœtida, in front of each nostril in turn, closing the other nostril with the finger. Ammonia or acetic acid must not be used, since these stimulate the sensory fibres of the fifth nerve and may produce a pungent sensation in the nose even when the sense of smell is lost.

In testing for anosmia we should inquire as to the sense of smell before the injury, and see that the nostrils are clear of blood-clot.

The anosmia complicating fracture of the anterior fossa is rarely permanent.

Ocular nerves :—The third, fourth, and sixth nerves.

All these nerves enter the orbit through the sphenoidal fissure, having previously traversed the walls of the cavernous sinus. The discussion of lesions of the ocular nerves from intra-cranial injuries does not fall within the scope of the present work. It may, however, be mentioned that intra-cranial hæmorrhages of traumatic origin are often associated with ocular palsies, the sixth nerve being the one most frequently affected.

The presence of ocular palsies is recognized by observing impairment of ocular movements, and also, more delicately, by noting the occurrence of diplopia. For the latter, it is essential that the visual acuity shall not be materially impaired by any lesion of the eye itself or of the optic nerve.

The fourth nerve supplies the superior oblique muscle, whose action upon the eye is to turn the anterior pole downwards and outwards ; the deficiency in movement of the eyeball on paralysis of the muscle is difficult to see, but the patient has diplopia when he gazes downwards or outwards. The patient feels giddy, especially when he looks downwards as in walking downstairs, and he habitually inclines his head forwards and towards the sound side.

The sixth nerve supplies the external rectus. Its paralysis is obvious since the affected eye cannot be turned outwards, but can be moved in all other directions.

With the exception of the superior oblique and the external rectus all the other ocular muscles are supplied by the **third nerve.** The third nerve also supplies the voluntary part of the levator palpebræ superioris ; it also contains fibres which through the ciliary ganglion and short ciliary nerves supply the sphincter pupillæ and the ciliary muscle.

In complete paralysis of the third nerve there is ptosis or drooping of the upper eyelid from paralysis of the levator palpebræ, with elevation of the eyebrow on the same side from over-action of the frontalis. In addition to this, there is external strabismus from the unopposed action of the external rectus, and there is inability to move the eye upwards, directly downwards, or directly inwards ; the eye can be moved slightly downwards and inwards by the superior oblique.

The pupil is dilated owing to the paralysis of the sphincter iridis, and does not contract to light or on attempted accommodation.

The Fifth, or Trigeminal Nerve.

This nerve has two roots, sensory and motor. The sensory root immediately distal to the gasserian ganglion divides into three divisions : ophthalmic, superior maxillary, and inferior maxillary ; the inferior maxillary is joined by the motor root of the fifth and then becomes a mixed nerve.

The ophthalmic division passes through the sphenoidal fissure into the orbit and supplies the eyeball and lachrymal gland, the conjunctiva (except that

of the lower lid), the skin of the forehead and scalp up to the vertex, the mesial part of the skin of the nose, and the mucous membrane of the upper part of the nasal cavity; it is joined at the gasserian ganglion by pupil-dilating fibres from the cervical sympathetic.

The superior maxillary division passes through the foramen rotundum across the spheno-maxillary fossa to the infra-orbital canal. It supplies the skin of the upper lip, the side of the nose, and adjacent part of the temple. It also supplies the conjunctiva of the lower lid, the upper teeth, and the mucous membrane of the following regions: the upper lip, the buccal cavity above the level of the angle of the mouth, the upper jaw including the alveolar margin and the hard palate, the soft palate and uvula, the naso-pharynx and middle ear, and the inferior nasal fossa.

The inferior maxillary division supplies the temporal, masseter, internal, and external pterygoid muscles, the tensor tympani, the mylo-hyoid and the anterior belly of the digastric.

When these fibres are implicated there is paralysis and atrophy of the masseter, temporal, and pterygoid muscles on the affected side. The weakness of the masseter and temporal is readily detected by placing the fingers over the affected muscles and making the patient tightly clench his teeth. The masseter and temporal muscles no longer harden and stand out as they ought to do. Weakness of the external pterygoid is dramatically shown by making

FIG. 37.—A SOLDIER WHO RECEIVED A GUNSHOT WOUND AT YPRES. Entrance just below right eye, exit in front of right tragus. The line marks out the areas anæsthetic to pin-prick and cotton-wool due to injury to the second and third divisions of the fifth nerve; the dotted areas are anæsthetic only to cotton-wool touches. There is also anæsthesia of the buccal cavity above the level of the angle of the mouth, of the right upper teeth, of the whole concavity of the right inferior turbinal and the anterior portion of its convexity.

the patient depress the lower jaw, when it at once swings over to the paralysed side, owing to the unopposed action of the opposite external pterygoid muscle. The jaw can no longer be voluntarily moved laterally towards the unaffected side.

The sensory distribution is to the posterior part of the temple and adjacent part of pinna, the anterior and upper wall of the external auditory meatus as far as and including the anterior part of the tympanic membrane, part of the cheek, the lower lip and chin, the lower teeth, the mucosa of the buccal cavity below the level of the angle of the mouth, the tongue (as far back as the circumvallate papillæ), the floor of the mouth, and the salivary glands.

Complete trigeminal paralysis from trauma is rare, and only occurs when the lesion is at or above the gasserian ganglion.

Partial lesions are relatively frequent. Thus a single division of the nerve may be implicated, or two divisions, especially the second and third, may be involved simultaneously by the same injury (Fig. 37).

Other cranial nerves are often injured along with the trigeminal, notably the ocular nerves, the facial, and the hypoglossal.

Seventh, or Facial Nerve.

This nerve has a motor and a sensory root, which meet at the geniculate ganglion.

The motor root arises in the lower part of the pons ; it forms a loop round the nucleus of the sixth nerve, then emerges at the lower border of the pons

immediately to the inner side of the auditory nerve ; between these two nerves is the sensory root of the seventh nerve. Both roots of the seventh nerve enter the internal auditory meatus and pass along the aqueduct of Fallopius, where they meet in the geniculate ganglion ; here they are joined by the great superficial petrosal nerve from Meckel's ganglion, and by the small superficial petrosal from the otic ganglion. As the facial nerve passes along the aqueduct it gives off a branch to the stapedius, and lower down the chorda tympani leaves it to join the lingual nerve ; it here runs in the inner wall of the tympanic cavity, covered by a very thin plate of bone, and here, too, it forms the floor of the aditus ad antrum.

The facial nerve emerges from the skull at the stylo-mastoid foramen and gives off branches to the occipital belly of the occipito-frontalis and the muscles of the pinna ; it then turns forwards, running in the substance of the parotid gland, and divides into branches which supply the stylo-hyoid and posterior belly of the digastric, and all the muscles of the face, excepting only the levator palpebræ superioris which is supplied by the third nerve.

The facial nerve may be injured during its passage through the temporal bone by suppuration in the middle ear, but much more often by operations on the mastoid antrum ; and after its emergence from the stylo-mastoid foramen by cold, by involvement in malignant growths of the parotid gland, and by operations on the parotid gland.

In warfare the nerve is liable to injury both during its course within the temporal bone and after its exit from the stylo-mastoid foramen, either alone or in conjunction with neighbouring nerves. The Fallopian aqueduct in the temporal bone may be injured directly by a projectile, or it may be implicated in a more widespread lesion of the skull, such as a fracture of the middle or posterior fossa. The facial nerve may also be damaged during its extra-cranial course through the parotid region, or in its terminal distribution in the facial muscles.

Symptoms.

Facial palsy is easily recognized.

In a complete case the face on the affected side is immobile, and its wrinkles become smoothed out. The lower lid droops, and, the punctum lachrymal not being kept in contact with the conjunctiva, tears trickle over the cheek instead of passing into the lachrymal duct. The eye is more widely open on the affected side, and cannot be closed ; on making the attempt the eyeball rolls upwards. The naso-labial fold is flattened, the angle of the mouth droops, and fluid dribbles from the affected side during drinking. During mastication, owing to paralysis of the buccinator, food tends to collect between the teeth and the cheek. The patient cannot purse up his lips, nor whistle. When the patient laughs or attempts to show his teeth, the mouth is drawn over to the non-paralysed side. In short, the face on the

paralysed side cannot be moved either voluntarily or emotionally.

If the lesion take place where the nerve is accompanied by the chorda tympani, i.e. in the Fallopian canal below the geniculate ganglion, in which part of its course it is intimately related to the inner wall of the tympanic cavity, we have, in addition, loss of taste in the anterior two-thirds of the tongue on the paralysed side.

Lesions of the facial nerve outside the skull produce no impairment of taste.

Extra-cranial lesions of the facial nerve are often partial, the lower fibres of the nerve being affected without the upper, or *vice versa.*

Figs. 38 and 39 are those of a soldier who sustained an extensive shrapnel wound of the face, extending from the upper part of the pinna to the angle of the mouth.

There is no cutaneous anæsthesia or analgesia.

There is complete paralysis of the right upper face, including the frontalis, orbicularis oculi, and zygomatici muscles. The depressor anguli oris acts well. To faradism the depressor anguli oris alone reacts.

The prognosis, as in the case of other nerves, is largely dependent upon the electrical reactions. If the reaction be normal, or slightly diminished to faradism and to galvanism, but with no polar changes, recovery may be expected within a month ; if partial reaction of degeneration be present, recovery may be looked for within two months ; if reaction of degeneration be present, no recovery of symptoms can be expected to appear before three months.

For treatment see p. 65. If no recovery manifests itself in six months, nerve anastomosis should be performed between the distal end of the facial nerve and the central end of the hypoglossal nerve.

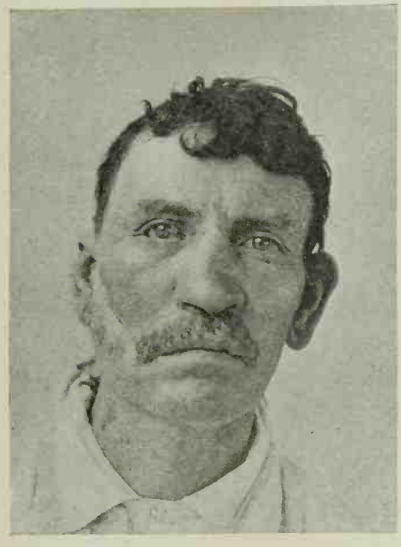


FIG. 38.—The face at rest, showing the right eye more widely open than the left, the furrows smoothed out of the right side of the face, and the right angle of the mouth drooping.

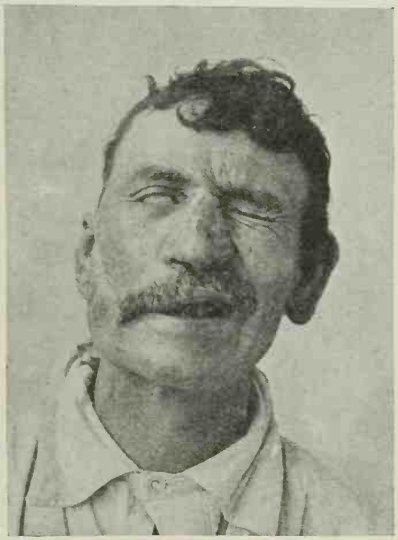


FIG. 39.—A forcible attempt to close both eyes. The right eye remains open and the eyeball is turned up under the upper lid; the mouth is drawn over to the non-paralysed side.

FIGS. 38 and 39.—A LACERATED SHRAPNEL WOUND OF THE RIGHT SIDE OF THE FACE, INJURING THE FACIAL NERVE.

The Eighth, or Auditory Nerve.

Injury to this nerve occasionally takes place in those fractures of the skull which pass through the petrous portion of the temporal bone, and as the nerve is here running with the facial nerve, the resulting deafness is in a large proportion of cases associated with facial paralysis. In testing such

cases for deafness, eliminate by visual examination the deafness due to the presence of blood or wax in the meatus; and note that in the deafness due to the affection of the internal ear or of the auditory nerve (so-called 'nerve deafness'), a tuning-fork applied to the skull is not heard on the affected side.

The Ninth, or Glosso-pharyngeal Nerve.

No case of isolated injury of this nerve has been recorded. Paralysis of the nerve causes anæsthesia of the back of the tongue and pharynx, difficulty in swallowing, and deficient taste in the posterior third of the tongue.

The Tenth, Vagus, or Pneumogastric Nerve.

This nerve has been injured in deep dissections of the neck. In the thorax it may be pressed upon by new growths or by aneurysms. Division of one nerve does not result in death, but its manipulation during operation may result in temporary cessation of pulse and respiration. Paralysis of the **recurrent laryngeal nerve** from pressure occurs in aneurysms of the aorta and thoracic new growths; division of the nerve is not uncommon, and usually occurs in operations for removal of a thyroid lobe, where it is cut or included in a ligature as it runs up behind the thyroid in the groove between the trachea and œsophagus. The way to avoid injury is to keep the wound dry by clamping vessels before they are cut, and by resisting the temptation to plunge with the artery-forceps if a vessel should bleed when

shelling out the lobe from its bed; and best of all, by keeping away from the nerve, by cutting through the posterior part of the gland and leaving behind that portion which covers in the space between trachea and œsophagus.

The vagus nerve, in addition to supplying inhibitory fibres to the heart and branches to the bronchial muscles, respiratory passages, œsophagus, stomach and intestines, contains motor fibres for the levator palati, and through its recurrent laryngeal branch supplies all the muscles of the larynx, with the exception of the crico-thyroid.

If the lesion be of the whole vagus nerve there is motor paralysis of the soft palate, with motor and sensory paralysis of the larynx.

To recognize unilateral paralysis of the palate, the patient is made to say 'Ah' whilst the uvula and soft palate are watched; normally the uvula and median raphé rise straight up, but if one side be paralysed they deviate to the healthy side.

Paralysis of the recurrent laryngeal nerve is easily diagnosed on making a laryngoscopical examination. The vocal cord on the affected side is found to be fixed in the cadaveric position, i.e. immobile, midway between abduction and adduction.

The Eleventh, or Spinal Accessory Nerve.

This is exclusively a motor nerve, and is distributed to the sterno-mastoid and to the upper and lower fibres of the trapezius (the middle fibres

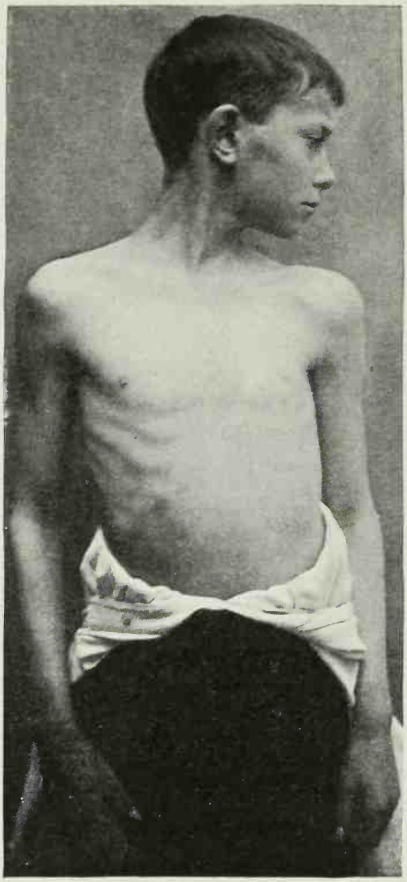


FIG. 40.—Shows the normal appearance of the right sterno-mastoid on rotating head to the left.

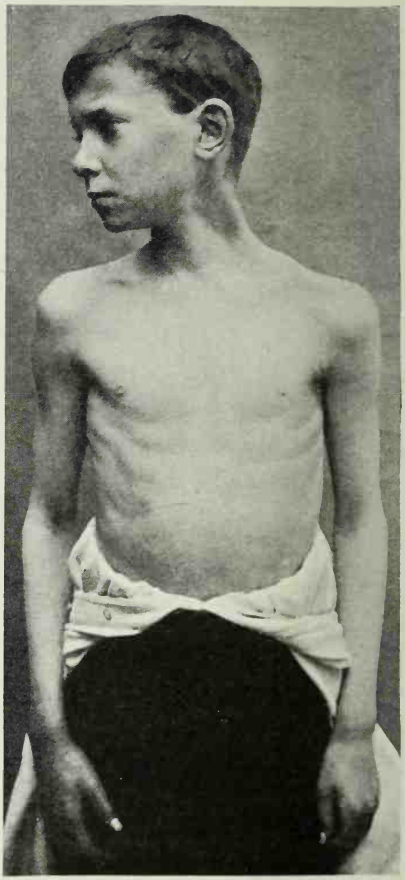


FIG. 41.—Shows absence of the left sterno-mastoid, but perfect movement of head to the right.

FIGS. 40 and 41.—PARALYSIS OF LEFT SPINAL ACCESSORY NERVE, FOLLOWING REMOVAL OF TUBERCULOUS GLANDS JUST BELOW THE LEFT MASTOID PROCESS.

are supplied by the third and fourth cervical nerves).

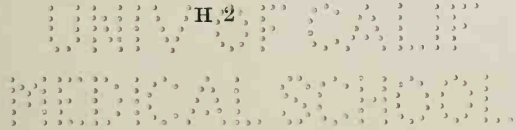
The spinal accessory may be injured in front of or behind the sterno-mastoid ; if in front, then the sterno-mastoid is paralysed, and more or less of the trapezius according to the amount of the muscle supplied by the cervical nerves ; if behind, then the sterno-mastoid escapes. This nerve is most commonly injured when tuberculous glands are being dissected out of the posterior triangle. Paralysis of the sterno-mastoid is evidenced by no deformity other than the loss of contour on that side of the neck. The head can be moved freely in all directions, but on rotating the head to the opposite side, or on depressing the head against resistance, the sterno-mastoid does not stand out as on the normal side. (Figs. 40 and 41.)

Paralysis and atrophy of the trapezius is evidenced by an alteration in the contour of the neck, and by displacement of the scapula (Fig. 42).

The alteration in contour is due to the levator anguli scapulæ, which, by the atrophy of the trapezius, is now permitted to display itself.

The scapula as a whole is displaced downwards and outwards, its upper end is rotated outwards and downwards, and its lower end upwards and inwards, so that the shoulder is dropped, and the inferior angle of the scapula lies nearer the mid-line than does the upper part of the vertebral border.

Fig. 42 is that of a soldier wounded by a rifle bullet. The entrance is one inch below and half an inch in front of



the right mastoid process; the exit is half an inch to the right side of the fourth cervical spine.

There is atrophy and paralysis of the right sterno-mastoid and right trapezius. The right rhomboids are subcutaneous.

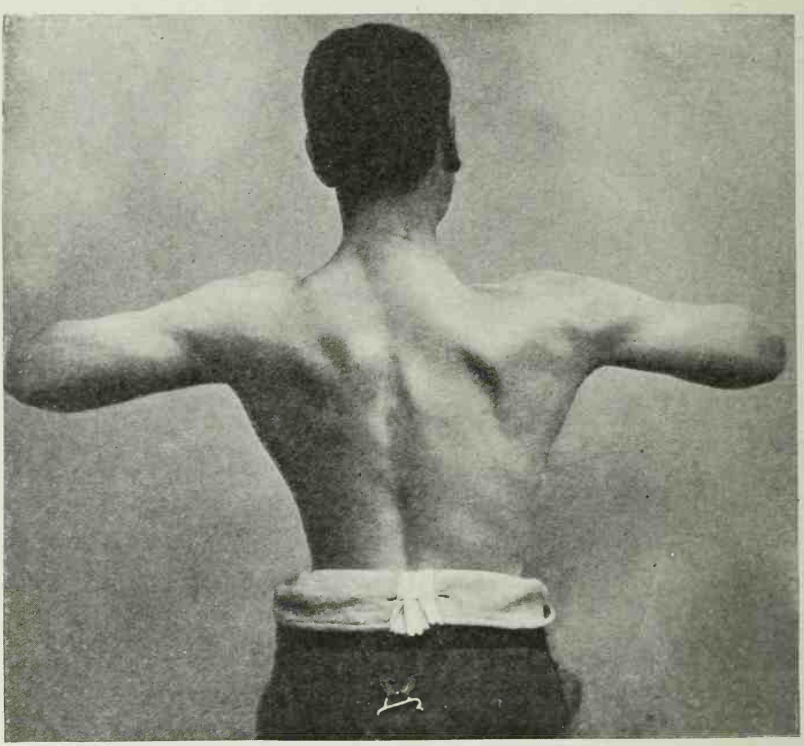


FIG. 42.—PARALYSIS OF THE RIGHT SPINAL ACCESSORY NERVE. Note the altered contour of the right side of the neck from wasting of the trapezius, and the slight 'winging' of the inferior angle of the right scapula.

The right scapula is displaced downwards and outwards, its lower end being tilted inwards and upwards.

There is no reaction to faradism in the sterno-mastoid or trapezius.

The Twelfth, or Hypoglossal Nerve.

The hypoglossal nerve supplies all the intrinsic muscles of one-half the tongue. Paralysis of one

hypoglossal results in that half of the tongue becoming atrophied; on protrusion of the tongue

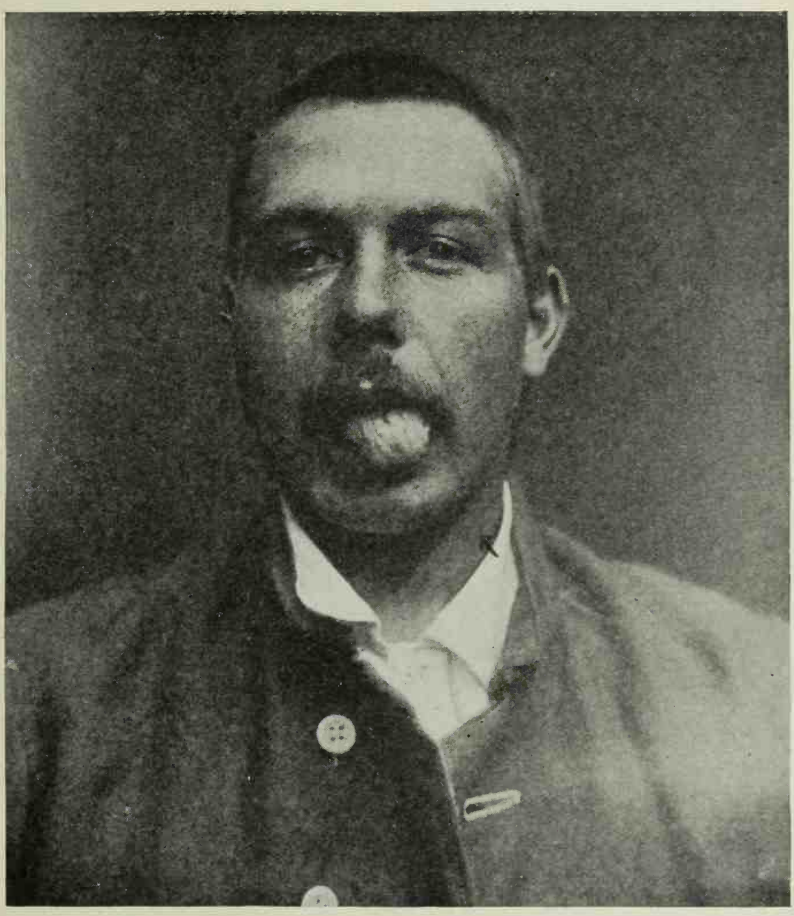


FIG. 43.—PARALYSIS OF THE LEFT HYPOGLOSSAL NERVE, AND OF THE RIGHT LINGUAL AND INFERIOR DENTAL NERVES—FOLLOWING A RIFLE-BULLET WOUND. The drainage tube is in the wound of entrance, and the arrow points to the scar of exit.

it points to the paralysed side. Immediately after division of the nerve the patient feels as if the paralysed half were a foreign body interfering

with mastication and articulation, but this sensation soon passes off. Sometimes vaso-motor changes are apparent, and the tongue is paler on the atrophied side; this is consequent on concomitant injury of the communicating branch from the cervical sympathetic.

Fig. 43 is that of a soldier shot by a rifle bullet. The entrance is in front of the right ear—the bullet penetrated the ascending ramus of the mandible; the exit is at the anterior border of the left sterno-mastoid on a level with the thyroid notch.

There is paralysis of the left hypoglossal nerve and the tongue deviates to the left on protrusion; there is also paralysis of the right lingual and inferior dental nerves with anæsthesia of the anterior two-thirds of the right half of the tongue, of the right inferior buccal cavity, and of the right lower teeth.

Cervical Sympathetic

The cervical sympathetic extends from the root of the neck behind the subclavian artery upwards to the base of the skull; it lies behind the common and internal carotid arteries. It may be injured by stab wounds, rifle and shrapnel bullet wounds, traction injuries of the brachial plexus, tumours at the root of the neck, and also in deep dissections in the neck necessitating interference with the internal jugular vein and carotid arteries.

Branches from the sympathetic supply—

1. Motor fibres to the pupil dilator.
2. Vasomotor fibres to the arteries of the head, neck, and arm.

3. The sweat glands of the head and neck, arm, and upper part of the trunk extending downwards to the third rib in front, and the spine of the scapula behind.

4. The non-striated part of the levator palpebræ, and the orbital muscle of Müller.

5. Secretory fibres to the submaxillary gland.

Paralysis of the cervical sympathetic is consequently evidenced by the following signs: the pupil is smaller than on the other side, it does not dilate when shaded, yet does contract to the stimulus of light and on convergence. The palpebral fissure is narrower, owing to the drooping of the upper eyelid; the patient can, however, voluntarily raise the lid to its full extent, the striated fibres of the levator palpebræ being supplied by the third nerve, so that the condition is not a true ptosis, but a 'pseudo-ptosis'. In addition, the affected eye is sunken in the orbit owing to paralysis of the non-striated muscle of Müller; to this the name of **enophthalmos** is given.

Fig. 44 relates to a soldier who was shot in January, 1915. The bullet entered the right side of the neck at the middle of the anterior edge of the sterno-mastoid; the exit was on the posterior aspect of the thorax, to the left of the second dorsal spine. In addition to paralysis of the brachial plexus (see p. 116), the patient had sustained a lesion of the right cervical sympathetic.

There was marked inequality of the pupils—in a dull light the right pupil measured 2·5 mm., the left 4 mm.; the right pupil reacted very slightly to darkness; pseudo-ptosis and enophthalmos were well marked. There was no difference in sweating on the two sides of the neck, trunk,

and limbs, so that all the fibres of the sympathetic had not been affected.

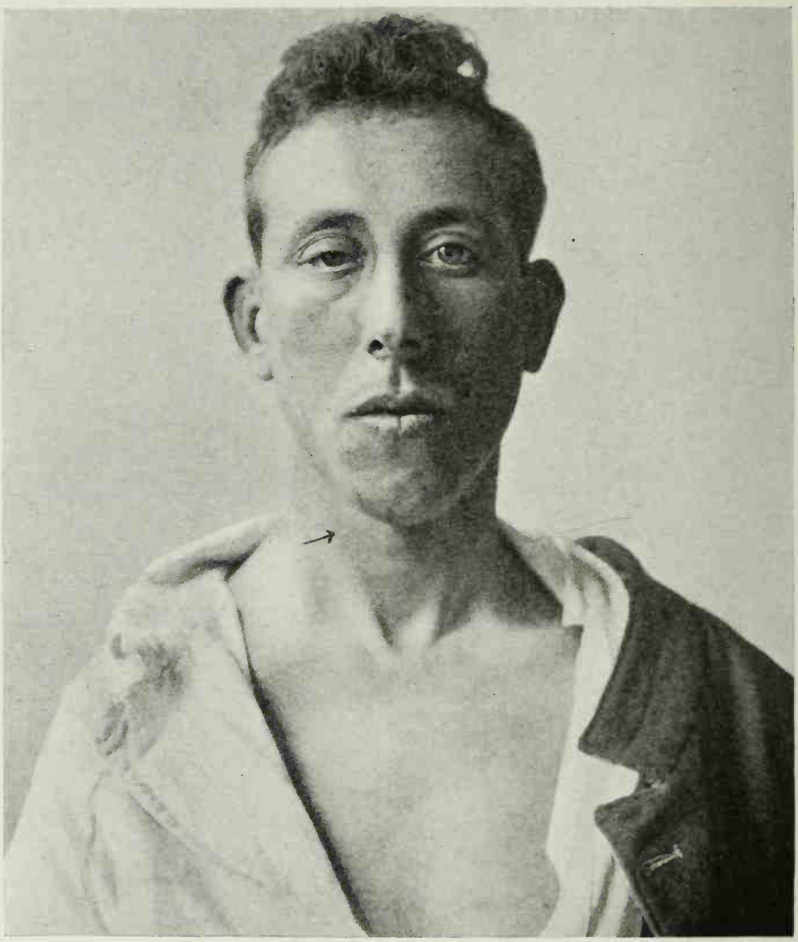


FIG. 44.—PARALYSIS OF THE RIGHT CERVICAL SYMPATHETIC. The arrow-head indicates the entrance wound; the exit is posterior, to the left of the second dorsal spine (see Fig. 66). Notice the pseudo-ptosis, and the enophthalmos. The right pupil measures 2·5 mm., the left 4 mm.

Irritation of the cervical sympathetic gives exactly the opposite signs: dilatation of the pupil, exophthalmos, and widening of the palpebral fissure.

This condition sometimes results from the pressure of an aneurysm or other tumour in the neck; later, if the pressure increases, the signs of stimulation may be replaced by those of paralysis.

Cervical Plexus

The superficial cutaneous branches of the cervical plexus arise from the anterior primary divisions of the upper four cervical nerves. From the second and third arise the small occipital, great auricular and superficial cervical nerves, and from the third and fourth arise the descending nerves, sternal, clavicular, and acromial. Their situation may be indicated by lines drawn from the mid-point of the posterior margin of the sterno-mastoid: the small occipital upwards along the posterior edge of the sterno-mastoid, the superficial cervical horizontally forwards, and the great auricular midway between these two. The direction of the others is sufficiently indicated by their names.

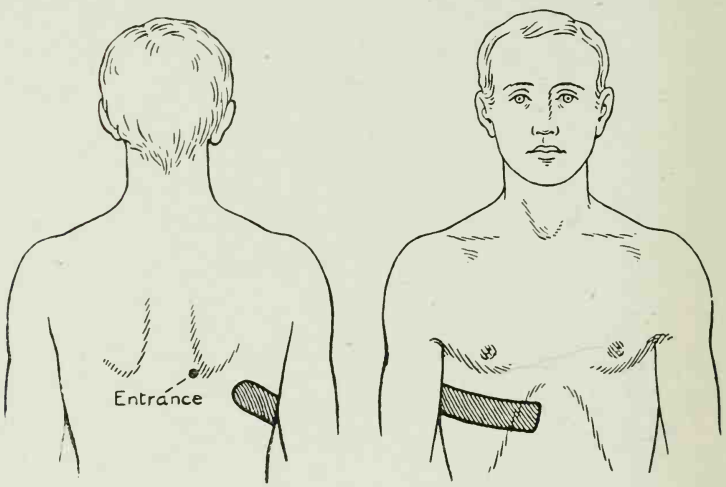
These nerves are most commonly injured as the result of operations in the posterior triangle of the neck, and particularly in extensive operations for the removal of tuberculous and of malignant glands.

These nerves, if divided during an operation, should be sutured on closing the wound, but failing this, careful co-aptation of the edges of the skin will usually result in their union.

Of the muscular branches the most important one is the **phrenic nerve.** This may be injured as it

runs downwards on the scalenus anticus to enter the thorax between the subclavian artery and vein. It is particularly liable to be injured when ligaturing this part of the subclavian artery.

Division of one phrenic nerve results in paralysis of the corresponding half of the diaphragm. The



FIGS. 45 A and B.—PARALYSIS OF THE SEVENTH RIGHT INTERCOSTAL NERVE AND RIGHT LONG SUBSCAPULAR NERVE. The bullet entered at the inferior angle of the right scapula ; the exit was near the apex of the right axilla. The shaded area is that anæsthetic to pin-prick. The right latissimus dorsi was paralysed.

inclusion of the nerve in a ligature has been known to occasion persistent coughing ; death with pulmonary symptoms has sometimes followed section of the nerve, but in the majority of cases the prognosis is good. If accidentally divided, the cut ends should be at once united.

Thoracic Nerves

The intercostal nerves are occasionally injured, either by extensive shell-wounds of the thoracic wall or by penetrating bullet-wounds of the thorax.

Figs. 45 A and B are those of an officer wounded in October, 1914, by a rifle bullet at two yards range.

The entrance wound is at the inferior angle of the right scapula ; the exit is in the mid-axillary line near the apex of the right axilla.

In addition to right hæmothorax, there was a zone of analgesia to pin-prick in the cutaneous area of the seventh dorsal segment on the lateral and anterior wall of the thorax.

The right latissimus dorsi did not contract voluntarily or on coughing, showing that in the passage of the bullet through the axilla the **long subscapular nerve** had been damaged.

CHAPTER VIII

THE BRACHIAL PLEXUS

THE **brachial plexus** is formed by the anterior primary divisions of the fifth, sixth, seventh, and eighth cervical nerves, and the greater part of the first dorsal nerve.

These nerves appear in the posterior triangle between the scalenus anticus and scalenus medius muscles, and their upper boundary in the neck is a line drawn from the centre of the sterno-mastoid muscle, at the level of the cricoid cartilage, to just outside the middle of the clavicle. Immediately after entering the posterior triangle the fifth and sixth unite, the seventh remains alone, and the eighth unites with the first dorsal, forming three primary cords; at the same time the four cervical nerves are dividing into anterior and posterior divisions. The secondary cords, usually spoken of as **the cords of the brachial plexus**, are named according to the position they occupy with regard to the axillary artery, and are formed thus: the anterior divisions of the fifth, sixth, and seventh unite to form the outer cord, the anterior divisions of the eighth and the whole of the first dorsal unite to form the inner cord, and the posterior divisions of the fifth, sixth, seventh, and eighth unite to form the

posterior cord. These nerves then break up into the various nerves of distribution.

The muscles supplied by, and the cutaneous distribution of these various nerves should be compared with the muscle-supply and cutaneous distribution of the anterior primary divisions from

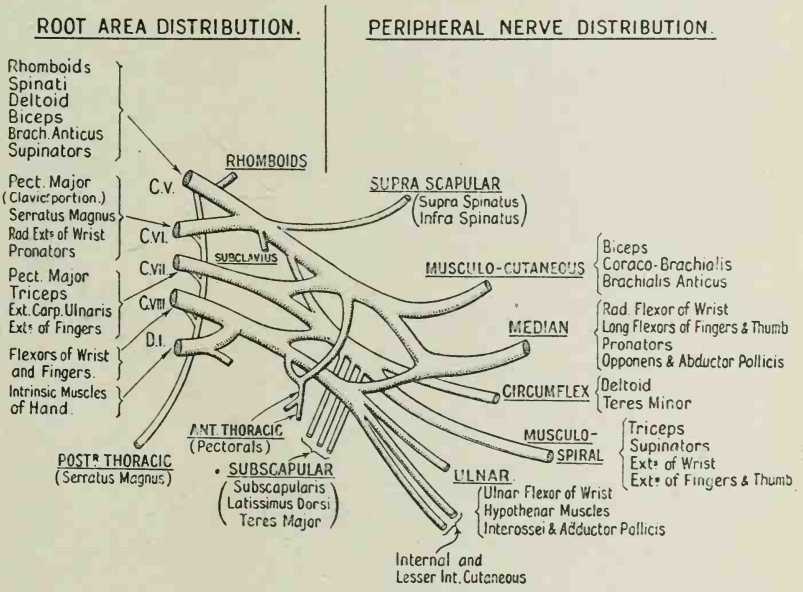


FIG. 46.—THE MOTOR-SUPPLY OF THE BRACHIAL PLEXUS, SHOWING BOTH ITS ROOT-AREA DISTRIBUTION AND ITS PERIPHERAL-NERVE DISTRIBUTION.

which they originate; the supply, both sensory and motor, of an anterior primary division is the same as that of the corresponding segment of the spinal cord from which it arises, and its distribution is according to root-areas. (See Figs. 46 and 47 A–D.)

It is a comparison of these two systems of distribution which enables one to diagnose the situation of the lesion.

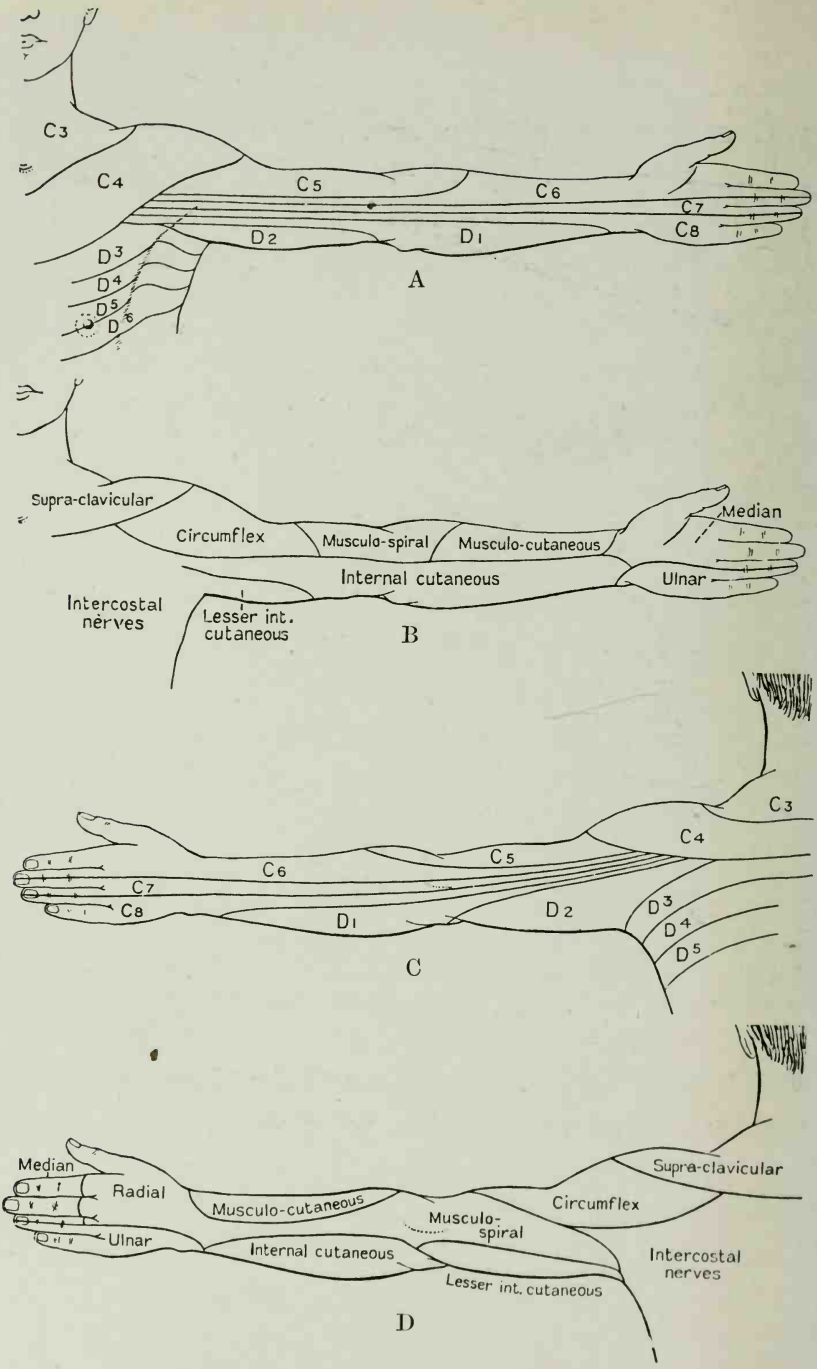


FIG. 47.—THE CUTANEOUS SENSORY SUPPLY OF THE UPPER LIMB. A and C show the areas supplied by the posterior roots; B and D the areas supplied by the peripheral nerves.

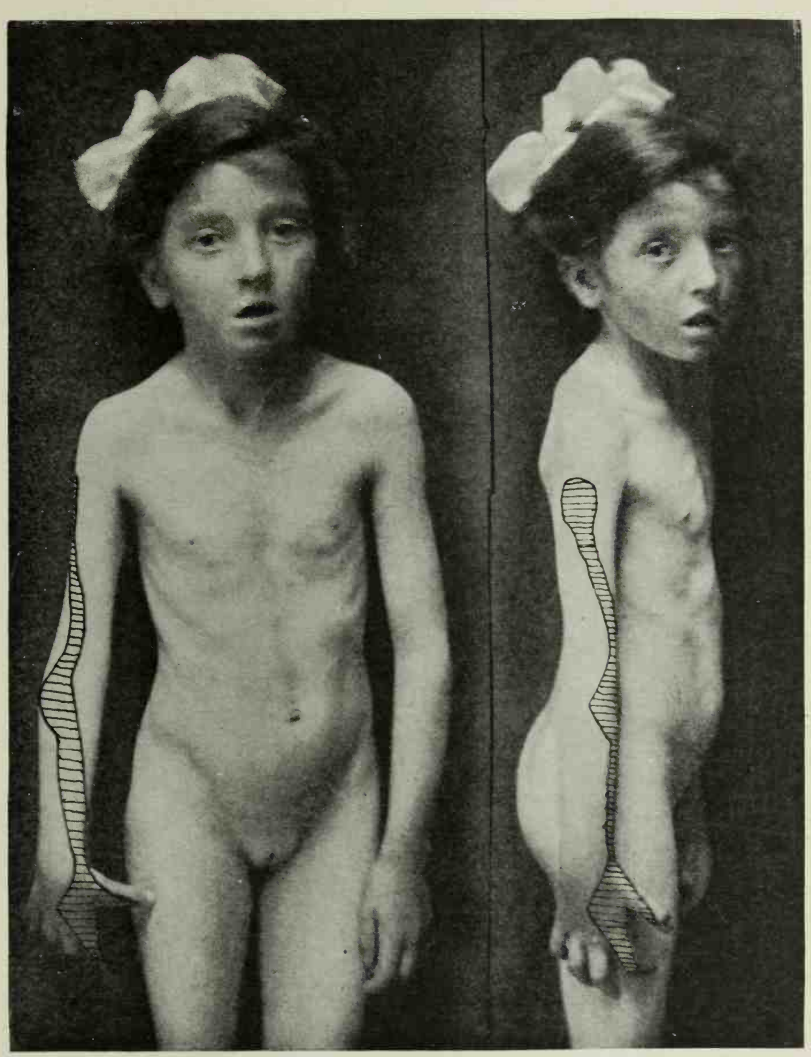


FIG. 48.—RUPTURE OF THE FIFTH AND PART OF THE SIXTH CERVICAL ROOT. Photographs of a child who, after a fall whereby she sustained a fracture of the right humerus above the elbow, developed wasting and weakness of the right arm. The area of anæsthesia is mapped out. The deltoid, supra-spinatus, infra-spinatus, biceps, and supinator longus are wasted; flexion of the elbow and supination of the forearm are impossible.

Fig. 48 is that of a child who subsequent to a fall which fractured her humerus, developed weakness and wasting of the right upper arm.

There is an area of anæsthesia down the outer side of the upper arm, forearm, and radial side of hand. There is wasting of the deltoid, supra-spinatus, infra-spinatus, biceps, and supinator longus.

Abduction of the arm is limited, but the deltoid contracts feebly; flexion of the elbow is impossible, extension is good; supination of the forearm is impossible. The movements of the wrist and fingers are practically normal.

The anæsthetic area corresponds to the peripheral distribution of the fifth and part of the sixth cervical root, and the muscles affected are all supplied by the fifth root.

Injuries to the brachial plexus are due to violence, direct or indirect.

Indirect injuries are always produced by overstretching, and are occasioned by violence applied more or less remotely from the plexus. They fall into two classes.

1. Violence applied so as to open out the angle between the head and neck and the shoulder of the same side (Fig. 49) produces a stretching of the brachial plexus in which the strain falls first on the fifth cervical nerve, then on the sixth, and so on, giving rise to **upper-arm paralysis** (Erb-Duchenne type). This lesion is most commonly produced by traction on the head in child-birth, or traction on the shoulder in breech presentations.

2. Violence applied so as to open out the angle between the arm and thorax (Fig. 50) produces a strain on the plexus which falls first on the eighth cervical and first dorsal, then on the seventh, and

so on, giving rise to lower-arm paralysis (Klumpke type). This occurs when a man falling from a height clutches at some means of safety, and the body weight is brought up with a jerk, widening the angle between the arm and thorax wall into a straight line, or, when in breech presentations the arm slips

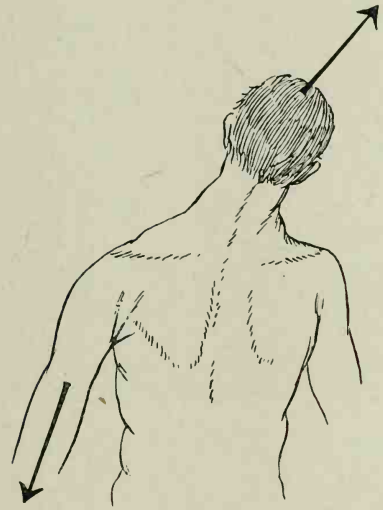


FIG. 49.—DIRECTION OF TRACTION-STRAIN PRODUCING LEFT UPPER-ARM PARALYSIS (Erb-Duchenne type).

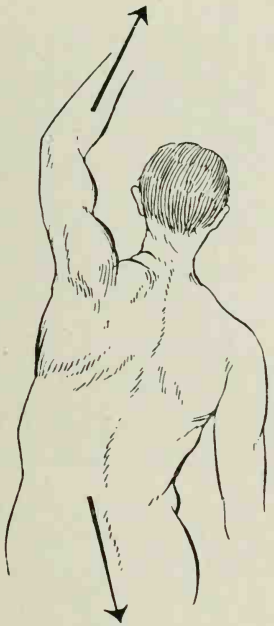


FIG. 50.—DIRECTION OF TRACTION-STRAIN PRODUCING LEFT LOWER-ARM PARALYSIS (Klumpke type).

up by the side of the after-coming head, and traction on the trunk is made.

The paralysis resulting from traction on the brachial plexus during child-birth is known as **brachial birth palsy**; this differs in no respect from other forms of traction injury.

Fig. 51 is that of a child seven days old. The birth was a breech presentation; it was very prolonged, and great difficulty was experienced in extracting the child.

On examination no abnormality was discovered save in the left upper limb. The arm was habitually adducted at the shoulder, extended at the elbow, and hyper-pronated.

There was total paralysis of the left deltoid, biceps, and supinators; all other muscles contracted normally.

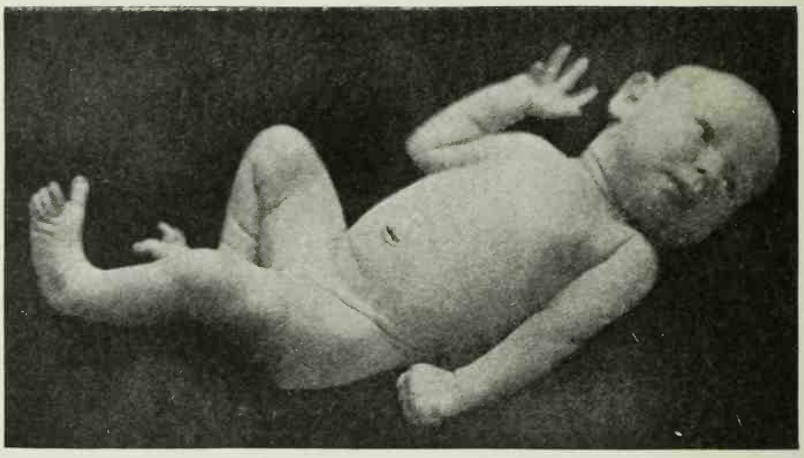


FIG. 51.—PARALYSIS OF THE FIFTH CERVICAL ROOT FOLLOWING DIFFICULT LABOUR IN A BREECH PRESENTATION. The arm is habitually adducted at the shoulder-joint, extended at the elbow, and hyper-pronated. The left deltoid, biceps, and supinators are paralysed; the deltoid and supinators do not react to faradism, the biceps reacts feebly; the other muscles contract and react normally.

To faradism the biceps reacted feebly, the deltoid and supinators not at all. All the muscles reacted to galvanism KCC = ACC.

We have here a lesion of the fifth cervical root.

Direct injuries.

In peace time the commonest cause of brachial plexus palsy in the adult is dislocation of the shoulder-joint, either from pressure of the displaced head of

the humerus (Fig. 58, p. 126), or from pressure of the heel in the axilla in attempting its reduction (this method should never be employed); it is sometimes produced by violence applied directly to the posterior triangle, or by the pressure of a cervical rib.

In the present war, direct injuries of the brachial plexus are the commonest of all nerve lesions, being produced by wounds of the neck, clavicle, thorax, and region of the shoulder-joint. Besides actual wounds of the plexus by projectiles or by bayonet-thrusts, it may be compressed by bony splinters from the clavicle, scapula, or humerus, or by a subclavian or axillary aneurysm.

Complicated injuries of the shoulder-girdle are often associated with extensive laceration of muscles, tendons, and ligaments; in such cases it requires considerable care to recognize what part, if any, of the disability is due to a lesion of the brachial plexus. Complete brachial palsy is rare; several cords may be simultaneously affected, but the commonest lesion is a partial one of a single cord.

Whilst this is true of the initial lesion, it must be remembered that the initial lesion is not the whole story; in the neighbourhood of the projectile track blood has been effused, adjoining portions of nerve structure have been infiltrated with blood, and neighbouring structures have been torn. By the time these cases come up for operation the blood has become organized, and the site of the lesion, the neighbouring nerve trunks, and the surrounding structures are wrapped in a mass of fibrous tissue.

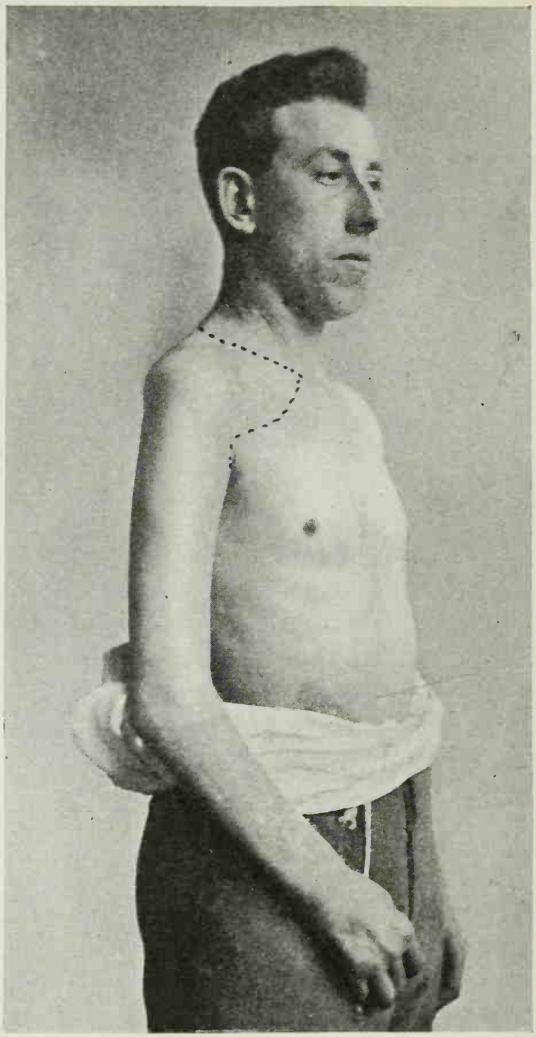


FIG. 52.—COMPLETE DIVISION OF THE RIGHT FIFTH CERVICAL ROOT, WITH INVOLVEMENT IN SCAR TISSUE OF THE OTHER ROOTS OF THE BRACHIAL PLEXUS, AND OF THE CERVICAL SYMPATHETIC. The arrow points to the entrance wound at the middle of the anterior edge of the sterno-mastoid; the exit wound is behind at the vertebral border of the left scapula on a level with the second dorsal spine. The whole arm and hand is anæsthetic to pin-prick and cotton-wool; the dotted line shows the upper limit. The pectoralis major, spinati, deltoid, triceps, biceps, extensors of wrist and fingers, flexors of wrist and fingers and the abductor and opponens pollicis are paralysed (slight flexion of little finger is possible), and do not react to faradism; the flexor carpi ulnaris, interossei, and adductor obliquus pollicis react to faradism. There is also right pseudo-ptosis with enophthalmos, and contraction of the right pupil (see Fig. 44).

Thus Fig. 52 relates to a soldier shot in January 1915. The entrance wound is on the right side of the neck at the middle of the anterior edge of the sterno-mastoid ; exit to left of the vertebral column on a level with the second dorsal spine. The photo shows the area which is insensitive to pin-pricks and cotton-wool.

The only muscles which reacted to faradism were the flexor carpi ulnaris, the interossei, and the adductor obliquus pollicis ; no other muscle reacted to faradism, not even the small muscles of the thumb supplied by the median, so that the eighth cervical and first dorsal did not entirely escape. The fifth, sixth, and seventh were entirely paralysed.

At the operation in June 1915 it was found that the fifth cervical nerve had been cut through about $\frac{3}{8}$ inch above its junction with the sixth. The fifth, sixth, and seventh were matted into one thick mass, and blended with the scalenus anticus muscle, which in this region was nearly all fibrous tissue (Fig. 24, p. 73). In this case too the cervical sympathetic was paralysed (see Fig. 44, p. 104).

The following case shows how extensive and complicated these lesions of the plexus can be :

Figs. 53 and 54 are those of a soldier wounded at Ypres by a rifle bullet in December 1914. The entrance wound is immediately above and just internal to the mid-point of the right clavicle ; the exit is situated at the axillary border of the right scapula, about midway between the angle of the scapula and the acromio-clavicular articulation.

June 1, 1915. The photographs show the area of anæsthesia to pin-pricks, also the wasted condition of the whole right arm, including the thenar and hypothenar eminences.

The following muscles react to faradism and voluntarily contract : rhomboids, supra- and infra-spinatus, deltoid, flexors of wrist, flexors of fingers, small muscles of thumb ; The following are paralysed : biceps, triceps, supinator longus, extensors of wrist, extensors of fingers, and interossei.

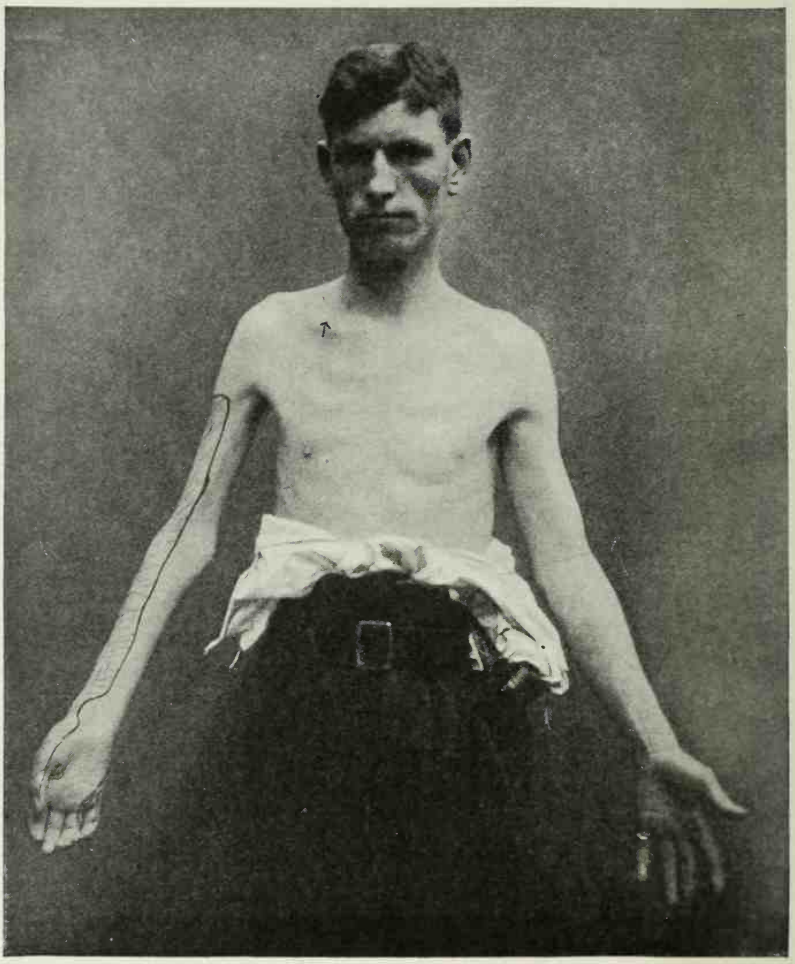


FIG. 53.—INCOMPLETE PARALYSIS OF THE WHOLE BRACHIAL PLEXUS. A rifle-bullet wound. The entrance is in front, the exit behind (Fig. 54); the arrows point to the scars. The line maps out the area anæsthetic to pin-prick. The following muscles were paralysed and did not react to faradism: biceps, triceps, supinator longus, extensors of wrist, extensors of fingers, and the interossei.

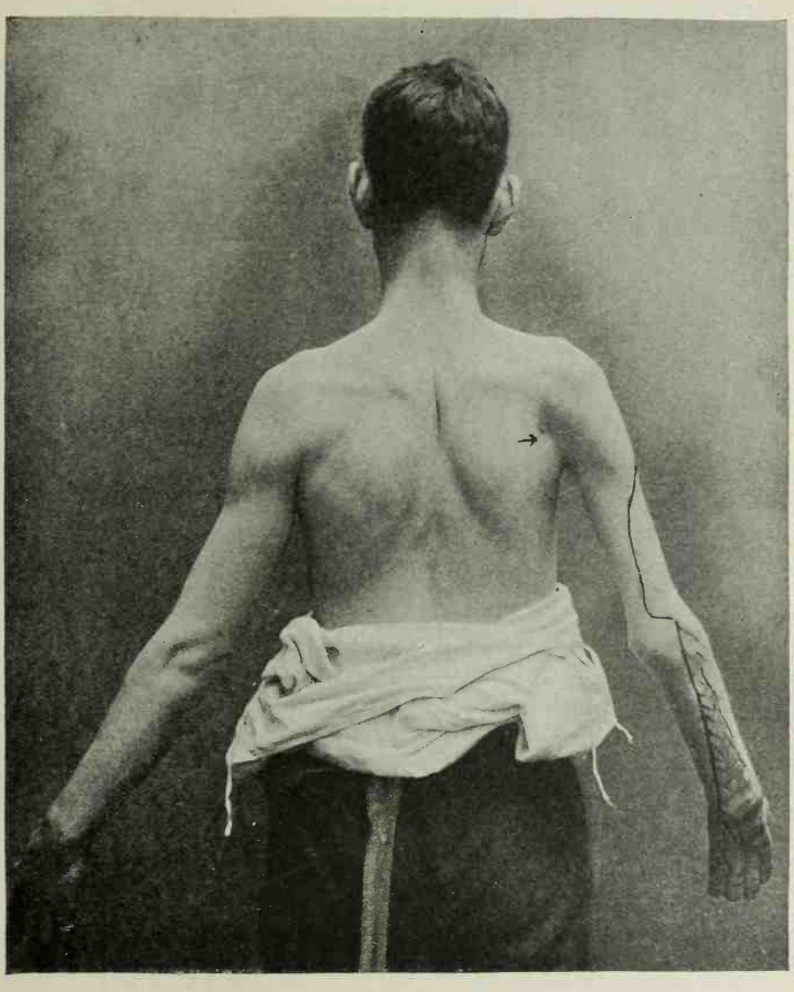


FIG. 54.—INCOMPLETE PARALYSIS OF THE WHOLE BRACHIAL PLEXUS.
(See Fig. 53.)

The diagnosis here is that of an incomplete paralysis of the whole brachial plexus. The sensory loss is that of the fifth, sixth, and eighth cervical roots; the distribution of the seventh to the anterior surface of the limb is preserved, that to the posterior surface is lost.

The motor distribution of the fifth cervical root is mostly preserved, and that of the eighth cervical and first dorsal; there is almost complete loss of the motor distribution of the seventh and partial loss of that of the sixth cervical root.

June 4, 1915. At the operation it was seen that the seventh nerve had a lesion on its inner side; it had been partially divided; there was some retraction and thickening of the proximal end of the nerve-lesion. Behind this and adherent to it the fifth and sixth swelled out into a bulbous enlargement which fused with a similar enlargement of the eighth cervical and first dorsal nerves.

This mass was hard to the touch, cut like fibrous tissue, and in places looked like fibrous tissue.

It appeared as though the bullet had hit the inner edge of the seventh cervical nerve, incising one half of it, and had then lacerated the contiguous edges of fifth and sixth cervical, and the eighth cervical and first dorsal nerves; the blood which was effused into and about them had apparently organized and caused them to become densely adherent to each other, and had given rise to a marked fibrosis of the nerves for about one inch of their length; there were also adhesions connecting this mass to the posterior surface of the clavicle. See Fig. 33, p. 79.

Inner cord of the brachial plexus.

Lesions of the inner cord are evidenced by a loss of sensation along the inner side of the arm, forearm

and hand, and by paralysis of all the intrinsic muscles of the hand, together with paralysis of some or all of the flexors of the wrist and fingers—usually it is only the flexors supplied by the ulnar nerve which are affected (flexor carpi ulnaris and inner half of flexor profundus digitorum).

Fig. 55 is that of a soldier shot in April 1915, at St. Julien, whilst building a parapet in a trench. The exit wound was in front of the right shoulder just internal to the neck of the humerus; the entrance was behind, about one inch to the left of the fourth dorsal spine; instantly he lost the use of his right arm and started spitting blood.

For the next month he was unable to use his arm, but later, with massage and electricity, he states that he could do anything with his shoulder, arm, and wrist, but could not close his fingers; with the right arm hanging down, the hand became dark blue in colour.

When seen on August 5, 1915, there was an area of anæsthesia on the inner side of arm and forearm. The following muscles contracted: deltoid, triceps, biceps, supinators, pronators, and extensors of wrist, fingers, and thumb. The following muscles did not contract and did not react to faradism: flexors of wrist, thumb and fingers, interossei, and short thumb muscles (abductor, adductor, and opponens). The palm was very wasted, and there was some contracture of the flexor tendons. On attempting to close the hand the unopposed extensors of the wrist forcibly dorsiflexed the wrist (Fig. 56).

The sensory loss is that of the first dorsal root; the motor loss is that of the eighth cervical and first dorsal roots, i.e. the inner cord of the brachial plexus.

At the operation on August 20, 1915, a sheet of dense fibrous tissue was found passing from the coracoid region to the under surface of the pectoralis minor; this was adherent to the sheath of the inner cord of the brachial

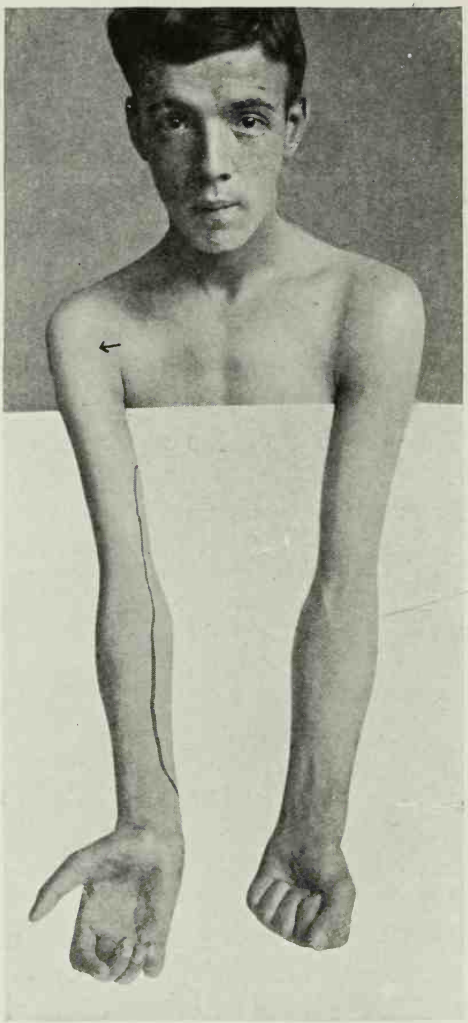


FIG. 55.—INJURY OF THE INNER CORD OF THE BRACHIAL PLEXUS. There is anæsthesia of the inner surface of arm and forearm corresponding to the cutaneous supply of the first dorsal root; the muscular loss corresponds to the eighth cervical and first dorsal roots (i. e. the inner cord), namely, paralysis of all the flexors of the wrist and fingers and of all the intrinsic muscles of the hand. Note the marked wasting of the palmar muscles, making apparent the long flexor tendons.

plexus. The deep surface of the cord, which was reddish, thickened, and nodular, was densely adherent to a mass of fibrous tissue, in which was the fibrosed and obliterated axillary artery (Fig. 30).

Fig. 57 is that of a soldier wounded by a rifle bullet. The bullet entered in the left pectoral

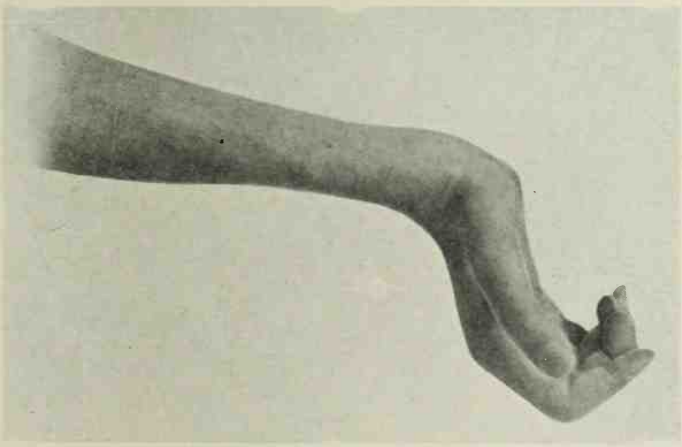


FIG. 56.—INJURY OF THE INNER CORD OF THE BRACHIAL PLEXUS, WITH PARALYSIS OF THE FLEXORS OF WRIST AND FINGERS, AND OF THE INTRINSIC MUSCLES OF THE HAND. There is some contraction and shortening of the long flexors. On making a forceful effort to clench the fist the unopposed extensors dorsiflex the wrist.

region, above and external to the nipple; its exit was at the upper end of the left posterior axillary fold.

Two weeks after the injury there was loss of sensation both protopathic and epicritic along the inner side of the left forearm and hand, front and back, including two and a half ulnar fingers in front and three fingers on the dorsum. Joint-sense and vibration-sense were lost in the two ulnar fingers and in their metacarpals.

The patient could not flex his fingers, oppose the thumb, or spread out his fingers; to faradism there was no response in the long flexors of the fingers, in the thenar or hypothenar muscles, or in the interossei; all the other muscles reacted normally. Exploration of the brachial plexus revealed no gross lesion of the

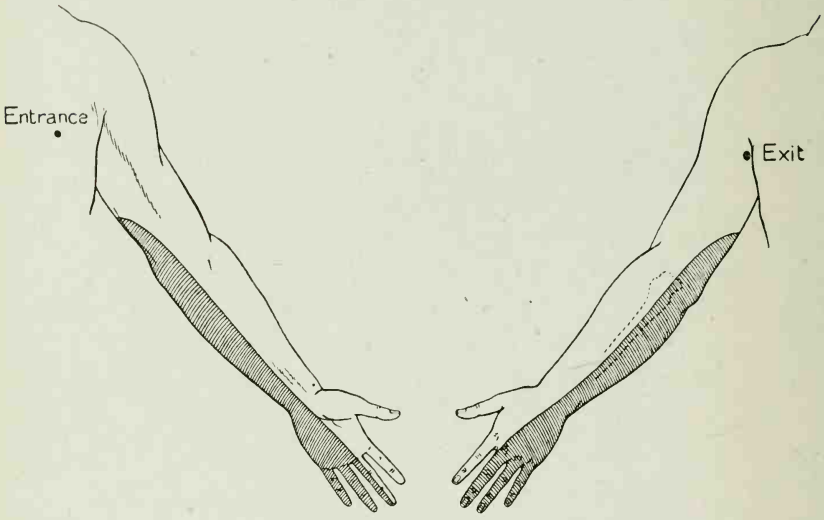


FIG. 57.—INJURY OF THE INNER CORD OF THE BRACHIAL PLEXUS. The shaded area is anæsthetic to pin-pricks and cotton-wool touches. There is paralysis of the flexors of the fingers, of the thenar and hypothenar muscles, and of the interossei. These muscles do not respond to faradic stimulation.

inner cord, but there was some scarring in the tissues on the outer side of the inner cord, along the line of the bullet track.

Posterior Cord of the Brachial Plexus.

Lesions of the posterior cord are evidenced by paralysis of the muscles supplied by the circumflex, musculo-spiral, and subscapular nerves, and by anæsthesia of the areas supplied by these nerves.

In a complete lesion the following muscles are paralysed: latissimus dorsi, subscapularis, teres major, deltoid, triceps, supinator longus, and the extensors of wrist and fingers.

A soldier, aged twenty-four, had a bullet-wound, entering half an inch to the right of the third thoracic spine. The exit-wound was an inch above the middle of the left clavicle, and an inch behind the sterno-mastoid. He had no hæmoptysis. The left upper limb at once fell powerless by his side. After the injury he developed intermittent pain along the flexor aspect of the left forearm from the elbow to the fingers, worst at nights.

On examination, a month later, a hard lump was felt above the left clavicle, in the position of the exit-wound, tender on pressure, not movable.

There was no cutaneous anæsthesia or analgesia of the left upper limb. Joint-sense was everywhere normal. The pectoralis major was powerful. The left deltoid was paretic but able to contract, though not enough to produce abduction of the shoulder. The biceps was powerful; the triceps was completely paralysed. There was also paralysis of the supinator longus and of the long extensors of the wrist, fingers, and thumb. The latissimus dorsi was paralysed and did not contract on coughing. All the other muscles of the limb were normal.

To faradism there was loss of reaction in the latissimus dorsi, triceps, supinator longus, and extensors of wrist, fingers, and thumb. All the other muscles reacted normally.

Lesion of the Posterior and Inner Cords of the Brachial Plexus.

Fig. 58 is that of a man who, when drunk, fell and dislocated his left shoulder; when seen the next day, eighteen hours afterwards, he was found to have a subcoracoid dislocation; this was easily reduced.

His left arm and hand were practically powerless; he could flex his elbow and, by means of the biceps, supinate his forearm.

One week later the faradic response was lost in the deltoid, triceps, supinator longus, extensors of

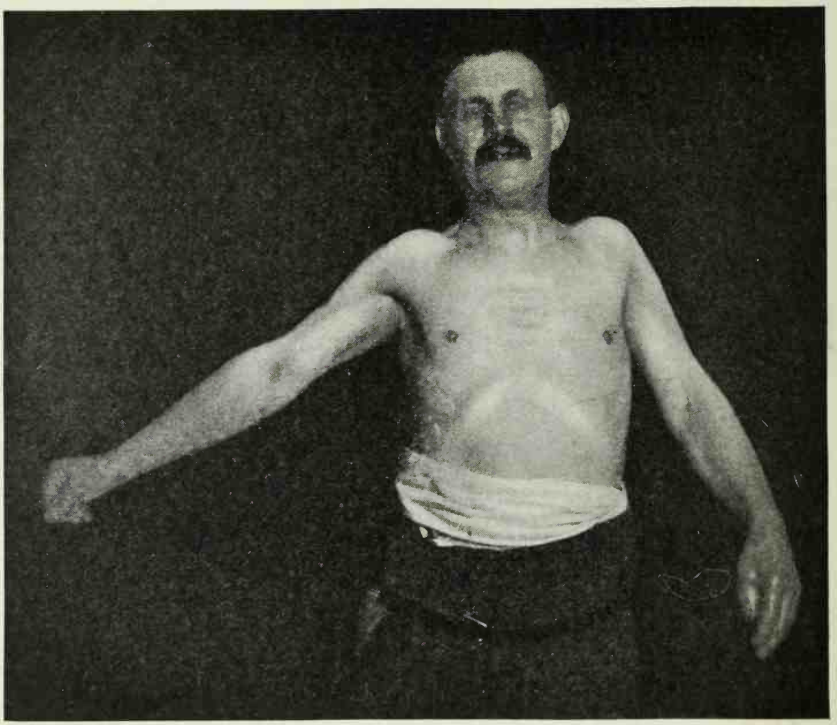


FIG. 58.—INJURY TO THE POSTERIOR AND INNER CORDS OF THE LEFT BRACHIAL PLEXUS, CAUSED BY A SUBCORACOID DISLOCATION OF THE HUMERUS.

wrist and fingers, and small muscles of the hand; there was a diminished response in the flexors of the forearm; the biceps response was normal to galvanism KCC > ACC, but the contractions were not brisk.

Three weeks after the injury the following was the response to galvanism: KCC > ACC in deltoid, supinator longus and interossei muscles; KCC = ACC in the extensors of wrist and thenar muscles; ACC > KCC in triceps, and extensor longus pollicis.

We have here a lesion of the posterior and inner cords of the brachial plexus.

Outer Cord of the Brachial Plexus.

Lesions of the outer cord result in paralysis of the muscles supplied by the musculo-cutaneous nerve and the median nerve (with the exception of the intrinsic muscles of the hand), i. e. there is paralysis of the biceps, coraco-brachialis, brachialis anticus, and the radial flexors of the fingers and wrist; there is also anæsthesia along the outer border of the forearm.

A sapper in the Australian engineers was wounded by a bullet which entered half an inch below the right clavicle, at a point half an inch internal to the coracoid process. The exit-wound, large and oblique, was in the left supra-spinous fossa, four inches long, with its upper end one inch to the right of the fourth thoracic spine. The patient had no hæmoptysis. The right upper limb at once dropped powerless. He felt as if he had received a violent blow in the back. The right upper limb at once became numb, especially in the fingers.

When examined, ten days after the injury, there was loss of sensation to touches and pin-pricks in nearly the whole upper limb, except along the inner side of the upper arm, and in the median and radial distributions in the hand. (See Fig. 59.) Joint-sense was lost at all the digits except the thumb; it was normal at the wrist, elbow, and shoulder.

All the muscles of the shoulder-joint were powerful. The biceps was totally paralysed, but the patient was still able to flex the elbow by means of the supinator longus. He could supinate the forearm, but could not pronate it beyond the mid-position. He could not flex the wrist, fingers, or thumb, but was able to extend them. All the intrinsic muscles of the hand were paralysed. To faradism there was loss of reaction in the long flexors of the wrist, fingers, and thumb,

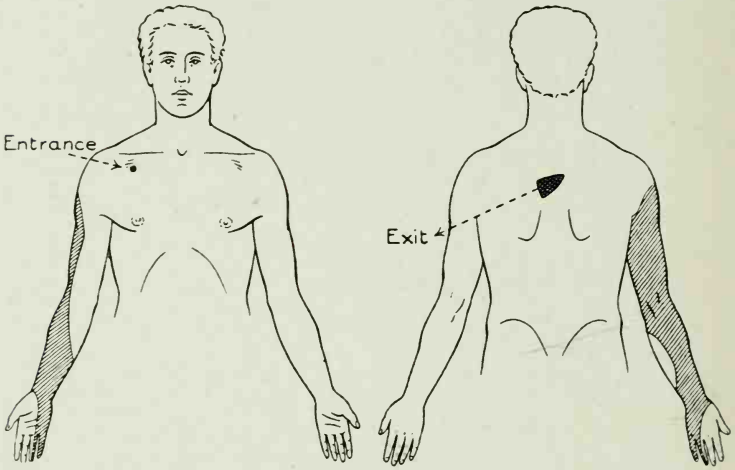


FIG. 59.—LESION OF BRACHIAL PLEXUS, MAINLY OUTER CORD. Anæsthesia of musculo-spiral, musculo-cutaneous, and ulnar areas.

also in the pronator muscles and in all the intrinsic hand muscles. The reaction in the biceps was feeble. There was a brisk response in the extensors of the wrist, fingers, and thumb, also in the supinator longus, triceps, and deltoid.

Hysterical Paralysis simulating a lesion of the Brachial Plexus.

A lesion of the brachial plexus may have a hysterical paralysis superadded, or a hysterical paralysis may closely simulate a plexus palsy, as in the following case :

An officer, aged 25, was wounded by a bullet in the region of the left shoulder. The entry wound was in front, just outside the surgical neck of the humerus; the exit was behind, and one inch below the entrance. No bone was fractured. The whole limb at once dropped powerless and 'dead'. When he came under observation four months later there was total anæsthesia and analgesia of the whole limb from the shoulder downwards, with the exception of a narrow strip running along the radial border of the forearm and hand. Joint-sense was absent at all joints from fingers to shoulders, and vibration-sense lost in the whole limb, including scapula. The shoulder, elbow, wrist, and most of the fingers were completely paralysed, and the limb dangled like a dead weight, flaccid and helpless (see Figs. 60 and 61). The only voluntary movement possible was feeble flexion of the thumb, index and middle fingers, and very feeble radial flexion of the wrist. The whole limb was slightly wasted. All the muscles, however, reacted normally, both to faradism and to galvanism. The diagnosis of hysterical paralysis was made.

Under nitrous oxide anæsthesia the patient moved all his fingers energetically, but nothing more. On coming round, before he recovered from the confusion of his anæsthetic, he was induced to move all the forearm muscles, and to flex the elbow repeatedly when aided by the stimulus of faradic shocks under the wrist.

In a few minutes, however, the paralytic symptoms reappeared as severely as ever, and the patient refused further treatment.

A few weeks later, when driving a motor-car along the street, using his non-paralysed hand only, he had to swerve suddenly to avoid a collision. Involuntarily he placed the paralysed limb on the steering wheel so as to assist his right hand. He then discovered that he was able to move the right limb well at all joints. When re-examined shortly afterwards, the anæsthesia was found to have disappeared, whilst motor power had returned.

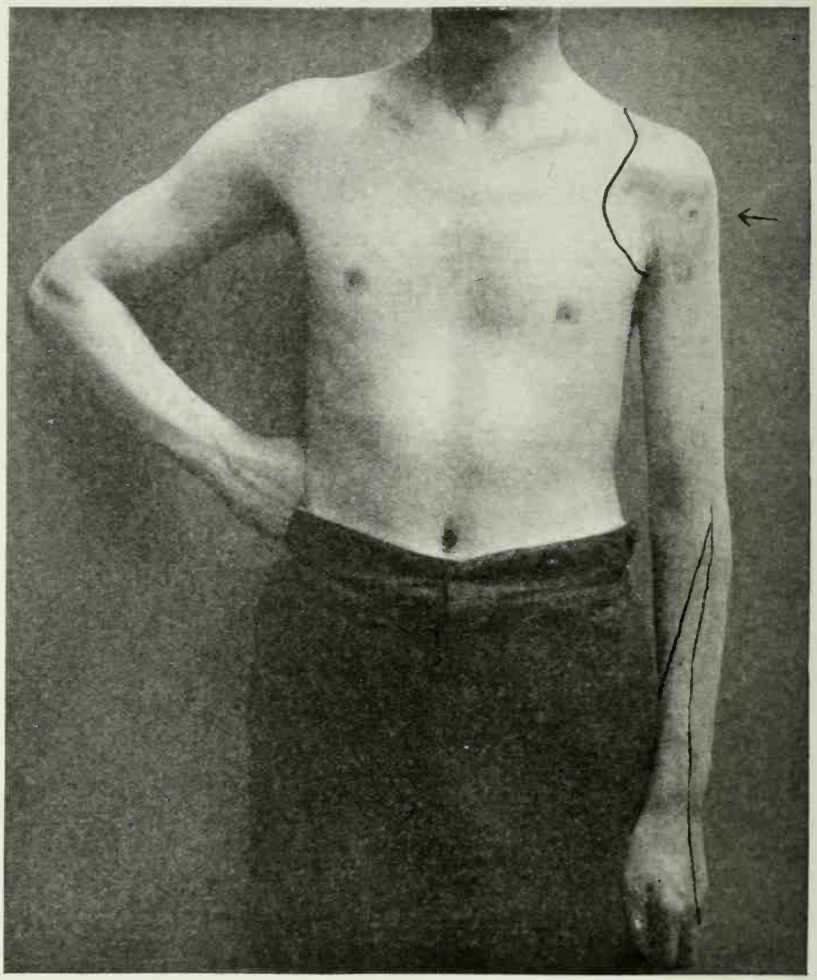


FIG. 60.—HYSTERICAL PARALYSIS OF THE LEFT UPPER LIMB. This immediately followed a gunshot wound. The arrow points to the entrance scar. The whole limb was anæsthetic save for the radial border of forearm, and radial three fingers. There was total paralysis, save feeble flexion of thumb, index and middle fingers, and very feeble radial flexion of wrist (i.e. of those muscles subjacent to the non-anæsthetic area).

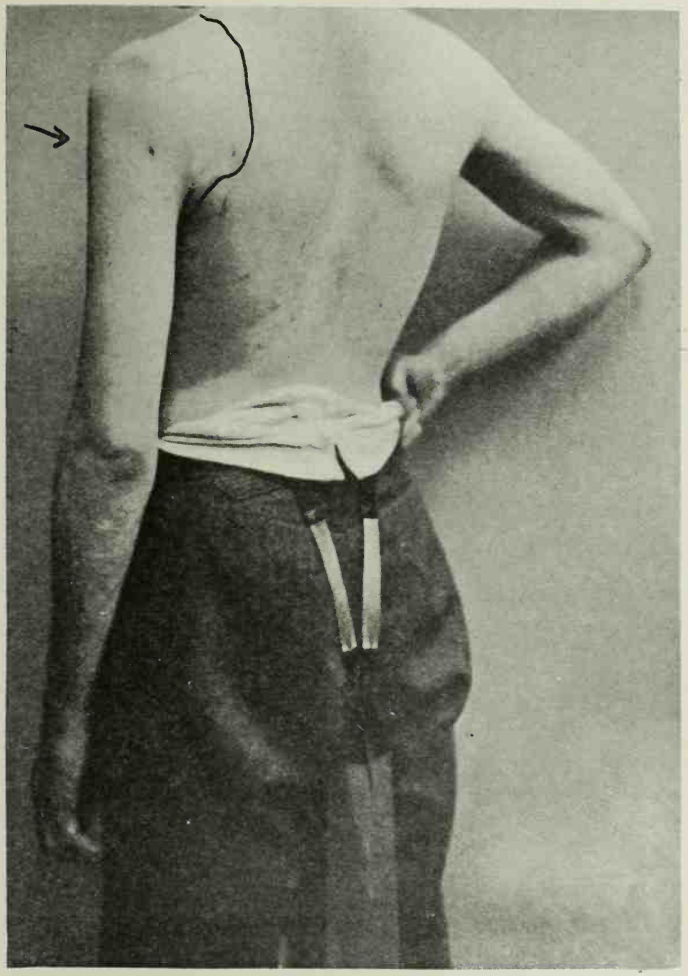


FIG. 61.—HYSTERICAL PARALYSIS OF THE LEFT UPPER ARM (see also Fig. 60). The arrow points to the scar of exit. The black line indicates the upper limit of anæsthesia.

Cervical rib.

Fibres from the eighth cervical and first dorsal nerves are occasionally injured by the pressure of a cervical rib. By 'cervical rib' is meant the exaggerated anterior tubercle of the transverse process of

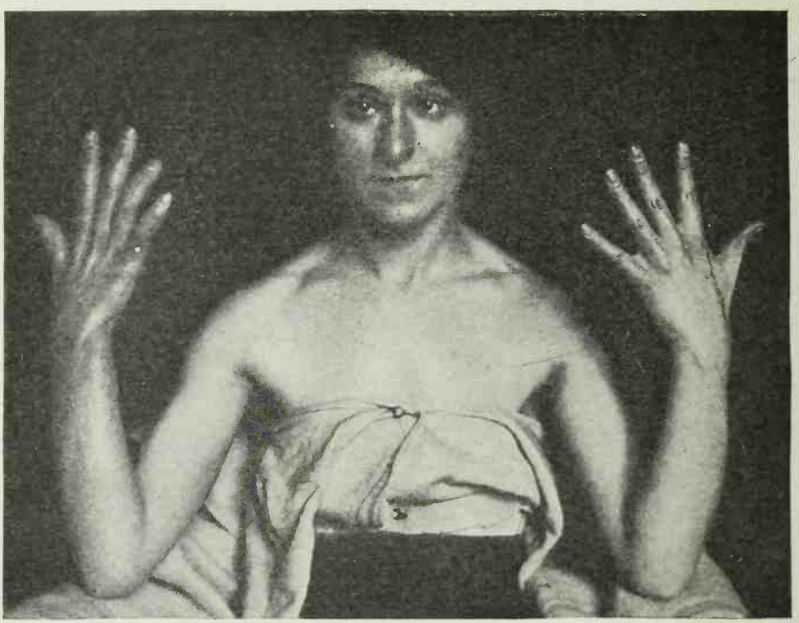


FIG. 62.—RIGHT-SIDED BRACHIAL PLEXUS INJURY FROM PRESSURE OF A CERVICAL RIB. The interossei and hypothenar muscles are wasted; there is hyper-extension of the two ulnar fingers at the metacarpo-phalangeal joints, and the patient complains of weakness in the right hand and tingling down the inner side of forearm and in the two ulnar fingers.

the seventh cervical vertebra, the extremity of which is connected by a fibrous band to the first rib or to the sternum.

Fig. 62 is the photograph of a woman, aged 28, who for six months had noticed gradual weakness

of the right hand, with tingling down the inner side of the forearm and in the two ulnar fingers. The patient presented no abnormality save in the right upper limb, and here there was no anæsthesia to cotton-wool, pin-pricks, or to temperature ; all the

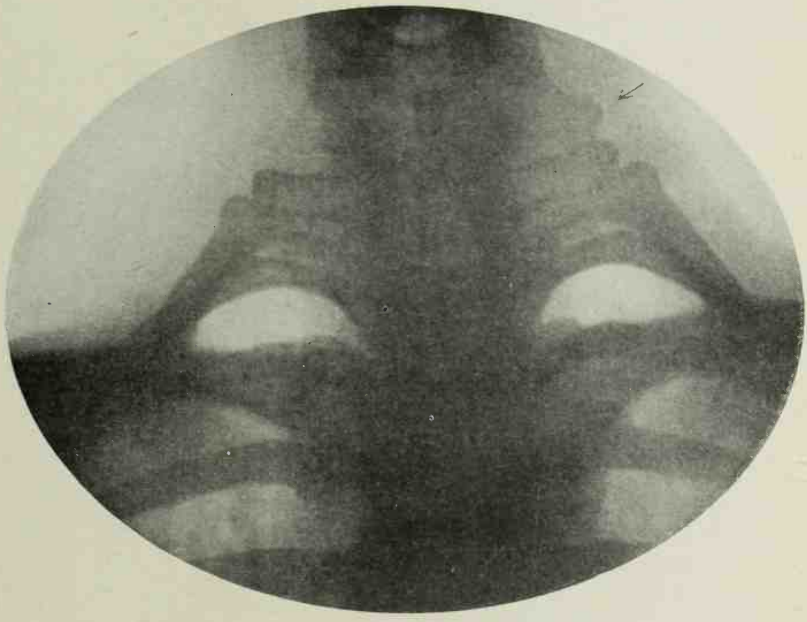


FIG. 63.—RADIOGRAM OF PATIENT SHOWN IN FIG. 62. The picture shows enlargement of the transverse processes of the seventh cervical vertebra, particularly well marked on the right side—to this the arrow points.

muscles were normal except the interossei and hypothenar muscles, which were wasted. There was slight hyper-extension of the two ulnar fingers at the metacarpo-phalangeal joints. There was slight wasting at the inner side of the thenar eminence, but all movements of the thumb could be freely executed. She could not spread out the fingers of

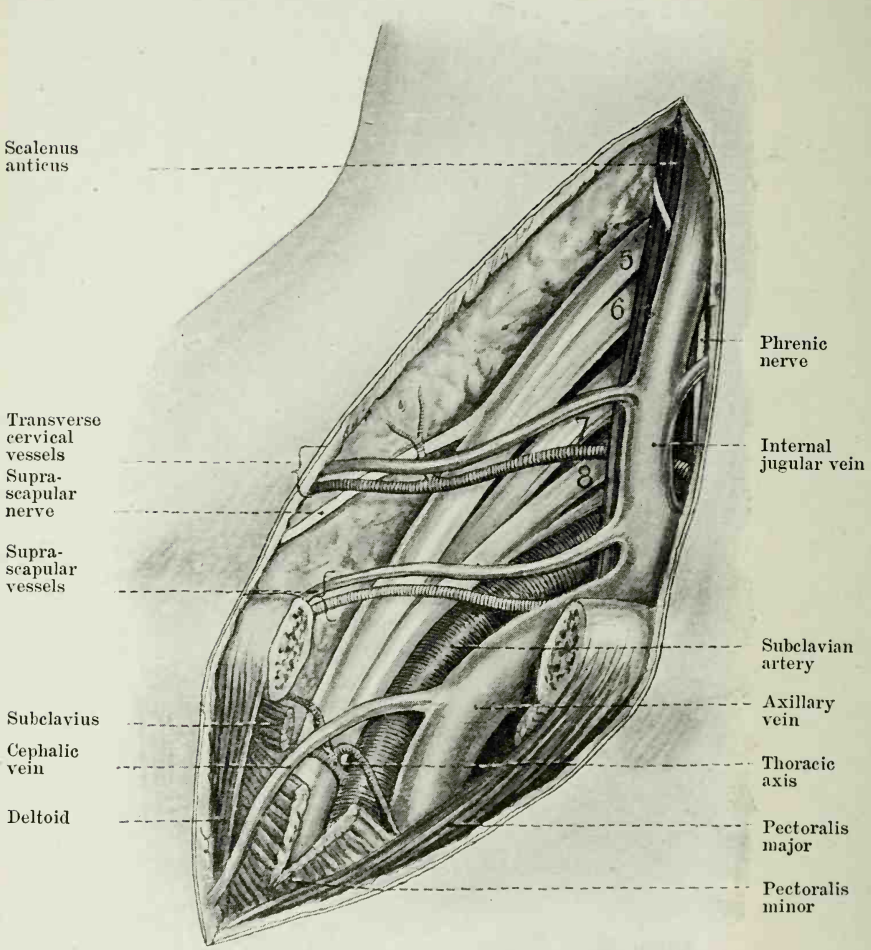


FIG. 64.—AN OPERATION TO EXPOSE THE RIGHT BRACHIAL PLEXUS. The incision commences at the middle of the posterior edge of the sterno-mastoid, and, having crossed the clavicle at the junction of the outer with the middle third, opens up the interval between the deltoid and pectoralis major muscles.

her right hand so well as she could those of her left.

The radiogram (Fig. 63) shows that the transverse

processes of the seventh cervical vertebra are enlarged, and particularly so on the right side; this was removed, and the posterior edge of the scalenus anticus muscle incised.

For the first few days the tingling, of which the patient had previously complained, was felt in the whole of the arm, but this entirely cleared up, including the abnormal sensations in the little and half the ring finger.

This patient later developed similar symptoms in the left hand from pressure of the left transverse process.

An operation to expose the brachial plexus.

An incision is made from the middle of the posterior edge of the sterno-mastoid to the junction of the middle and outer thirds of the clavicle. On opening the deep fascia in the same line the transverse cervical vessels will be seen; divide these between two clamps.

Near the upper end of the wound seek the posterior edge of the scalenus anticus, and here will be found the junction of the fifth with the sixth cervical nerve. Follow this down, and look out for the suprascapular nerve which comes off from the outer edge of the conjoined fifth and sixth nerves immediately below their junction. Below and internal to this nerve-trunk the seventh nerve will be found; and below and internal to this, the eighth.

Lower still and deeper, i.e. farther from the surface, the first dorsal nerve will be found, posterior to the subclavian artery.

To get a satisfactory exposure of the first dorsal nerve, prolong the incision downwards and outwards, in the interval between the deltoid and pectoralis major muscles, and cut through the clavicle. Cut through the periosteum of

the clavicle on its superior and anterior surfaces ; with an aneurysm needle separate the periosteum posteriorly. As this is being done, working from below, keep the needle closely hugging the bone ; when the eye of the needle appears above, pass a silk ligature through it and withdraw the needle. The silk is now tied to the end of a Gigli saw, and by means of this the saw is passed behind the bone. Saw through the clavicle, and cut through the periosteum, subclavius muscle, and costo-coracoid membrane. If these have not been encountered before, now clamp the supra-scapular vessels in two places, and divide between. If still the lesion be insufficiently exposed, incise the upper margin of the pectoralis minor, or cut right through it.

The exposure thus gained in the various stages is all one can desire (see Fig. 64).

At the end of the operation suture the pectoralis minor muscle and wire the cut ends of the clavicle.

THE POSTERIOR THORACIC NERVE

This nerve arises from the fifth, sixth, and seventh cervical nerves near the intervertebral foramina, and pierces the scalenus medius as two trunks, the lower being the branch from the seventh cervical nerve ; it runs down the side of the neck behind the brachial plexus, and enters the axilla between the upper edge of the serratus magnus and the axillary artery. It is a purely motor nerve, and supplies the serratus magnus muscle.

The nerve may be injured from violence applied to the supraclavicular region, or by wounds in the axilla, as in dissection of the axilla when operating for malignant disease of the breast. When injured in the supraclavicular region it is almost always

associated with lesions of other nerves, both of the brachial and cervical plexuses, so that the paralysis of the serratus magnus is generally associated with paralysis of the trapezius, rhomboids, spinati, &c.

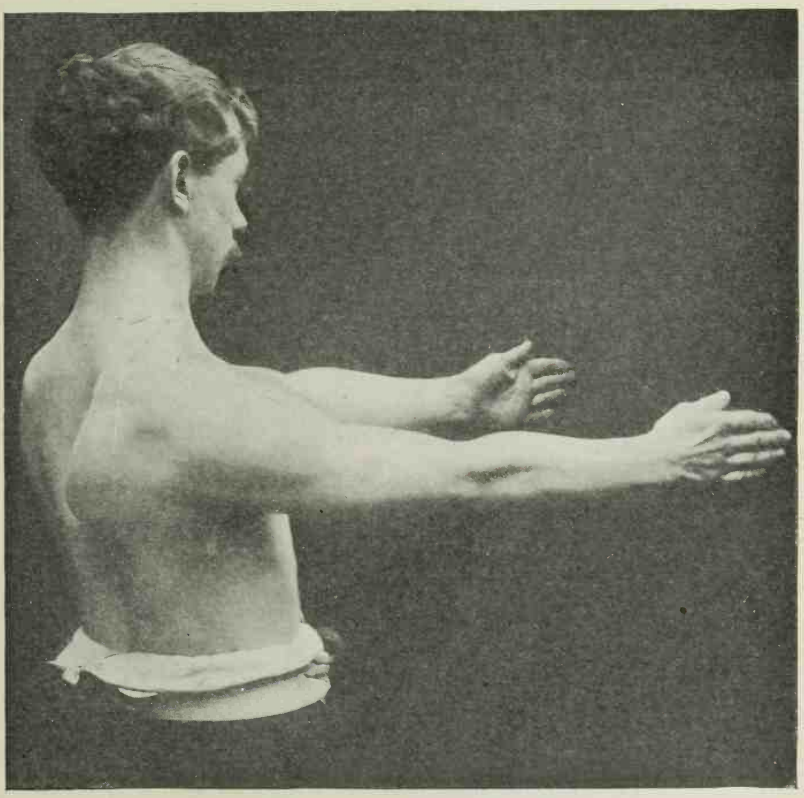


FIG. 65.—PARALYSIS OF THE RIGHT SERRATUS MAGNUS MUSCLE (posterior thoracic nerve), showing the marked protrusion of the inferior angle of the scapula when the arm is held horizontally in front of the body.

When the serratus magnus is the only muscle paralysed, the affection of the posterior thoracic nerve is generally occasioned by 'cold' or some toxic infection (Fig. 65).

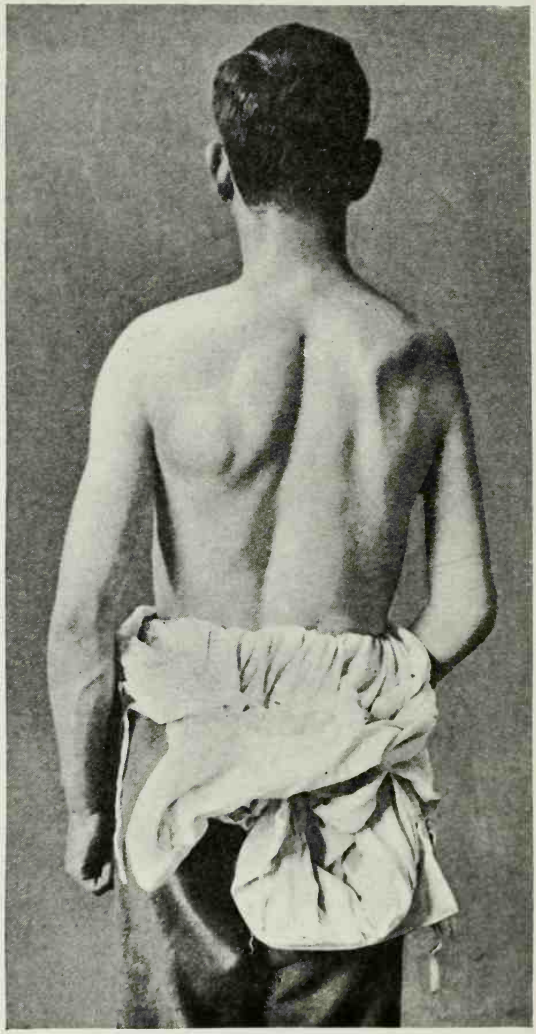


FIG. 66.—PARALYSIS AND WASTING OF THE RIGHT DELTOID AND SPINATI MUSCLES following a gunshot wound of the right side of the neck, which completely divided the fifth cervical root.

The serratus magnus keeps the scapula closely applied to the chest wall, and can advance the scapula towards the front; it also gives the deltoid and other upper-arm muscles a fixed base from which to work. When paralysed, with the arm hanging loosely at rest, there may be no obvious deformity, but on extending the arm horizontally in front of the body the scapula is thrust backwards away from the chest wall and becomes 'winged', and this is still more marked if a forward pushing movement is attempted. Where paralysis of the serratus magnus is associated with paralysis of the lower fibres of the trapezius, the deformity is more marked.

THE CIRCUMFLEX NERVE

The circumflex nerve is seldom injured in projectile wounds; in our own list of over three hundred nerve injuries this nerve is not found.

The circumflex nerve as it winds round the neck of the humerus may be injured in fractures of the neck of the humerus, in subglenoid dislocations, and from the pressure of a crutch.

Symptoms. There is paralysis and wasting of the deltoid muscle, with inability to abduct the arm.

In addition, there is a patch of cutaneous anæsthesia over the central portion of the muscle.

Paralysis of the deltoid is, however, much more commonly dependent on lesions of the fifth cervical root, and it is then associated with paralysis of the spinati and other muscles (Fig. 66).

CHAPTER IX

THE ULNAR NERVE

The ulnar nerve arises from the inner cord of the brachial plexus, and is derived from the eighth cervical and first dorsal nerves.

In the axilla it lies between the axillary artery and the vein, and in the upper half of the arm it lies on the inner side of the brachial artery; in the lower half it inclines away from the artery to the inner side of the limb and, passing through the internal inter-muscular septum, reaches the groove between the internal condyle of the humerus and the olecranon process; entering the forearm between the two heads of the flexor carpi ulnaris, it runs beneath this muscle until it reaches the pisiform bone; here it pierces the deep fascia and enters the hand superficial to the anterior annular ligament.

It gives off no branches in the arm, but in the forearm it supplies the flexor carpi ulnaris and the inner half of the flexor profundus digitorum. In the middle third of the forearm the ulnar nerve gives off a dorsal cutaneous branch which supplies the skin over the ulnar side of the back of the wrist, and the dorsum of the proximal phalanges of the little and half the ring fingers, and in the lower third of the arm a palmar cutaneous branch which supplies

the skin over the hypothenar eminence and the hollow of the palm.

In the hand the nerve supplies the palmaris brevis muscle and then divides into superficial and deep branches; the superficial branch supplies the skin over the palmar surface of the little and half the ring fingers, and the dorsal surface of the two terminal phalanges of the little and half the ring fingers; the deep branch supplies all the muscles of the hypothenar eminence (the abductor, flexor brevis, and opponens minimi digiti), all the interossei, the two inner lumbricales, the adductor obliquus, adductor transversus, and the deep part of the flexor brevis pollicis.

In civil life the ulnar nerve is most commonly injured in penetrating wounds about the wrist-joint; it is sometimes injured at the elbow as it passes behind the internal condyle, either by a direct blow or accompanying fractures and dislocations.

In warfare, injury of the ulnar nerve by penetrating wounds is common in every part of its course.

Symptoms of Ulnar Nerve Paralysis.

Sensory. In total ulnar palsy there is loss of cutaneous sensation in the little finger and the ulnar half of the ring finger and the corresponding part of the hand, front and back, not extending above the wrist. The protopathic loss is less than the epicritic, and loss of sensation to deep pressure is limited to the little finger (see Fig. 67). If the nerve is divided after it has given off its dorsal cutaneous branch, loss of sensibility will be confined

to the palmar surface of the hand and fingers, and the dorsal surface of the two terminal phalanges;

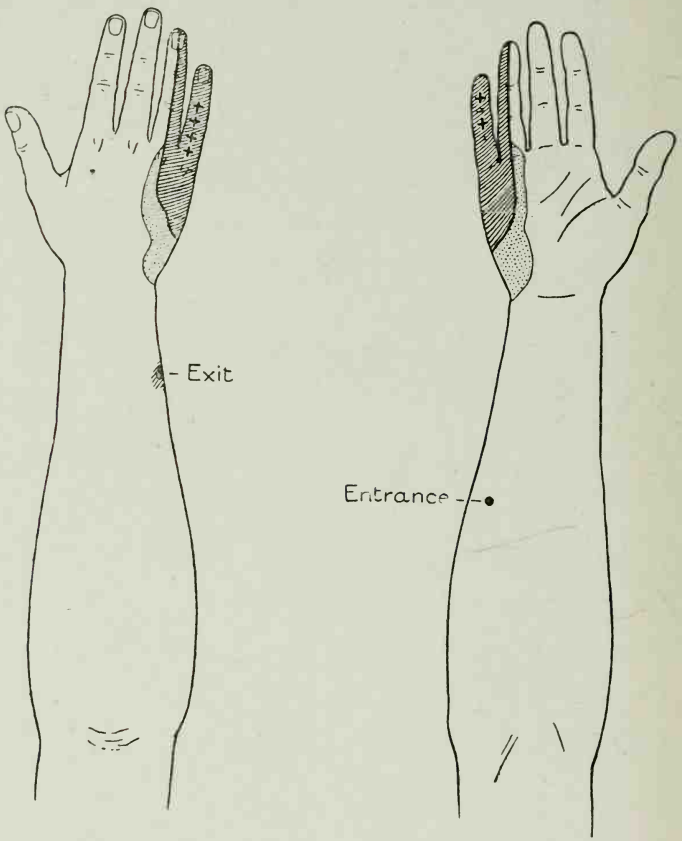


FIG. 67.—CUTANEOUS SUPPLY OF THE ULNAR NERVE. The shaded area is that insensitive to pin-pricks (protopathic loss). The dotted area is the additional area insensitive to cotton-wool touches (epicritic loss). The crosses indicate the region where there is loss of sensation to deep pressure. Joint-sense is lost in the little finger and vibration-sense in the two ulnar fingers. The ulnar nerve and its dorsal cutaneous branch were embedded in dense scar tissue.

if the division be lower still, after the nerve has given off its last cutaneous branch, there will be no area of anæsthesia (see Fig. 68).

Pain at the moment of injury is usually severe, shooting down the limb to the two ulnar fingers. This pain may persist for a considerable time, but

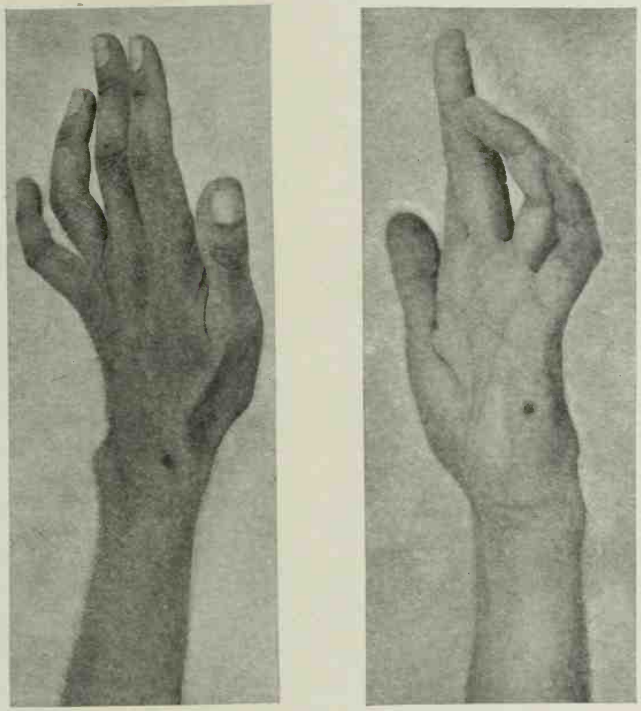


FIG. 68.—BULLET WOUND OF THE DEEP BRANCH OF THE ULNAR NERVE ACCOMPANIED BY NO SENSORY LOSS, AND WITH JOINT-SENSE NORMAL IN ALL JOINTS. Entrance-wound at ulnar border of hand, two inches below the wrist. Exit-wound on dorsum, the bullet having traversed the carpus. Note the position of the two ulnar fingers: hyper-extension at the metacarpo-phalangeal joints and semi-flexion at the interphalangeal joints.

compared with median palsy, pain is not an outstanding feature.

Motor. Owing to the paralysis of the interossei, whose action is to flex the metacarpo-phalangeal and extend the interphalangeal joints, the metacarpo-

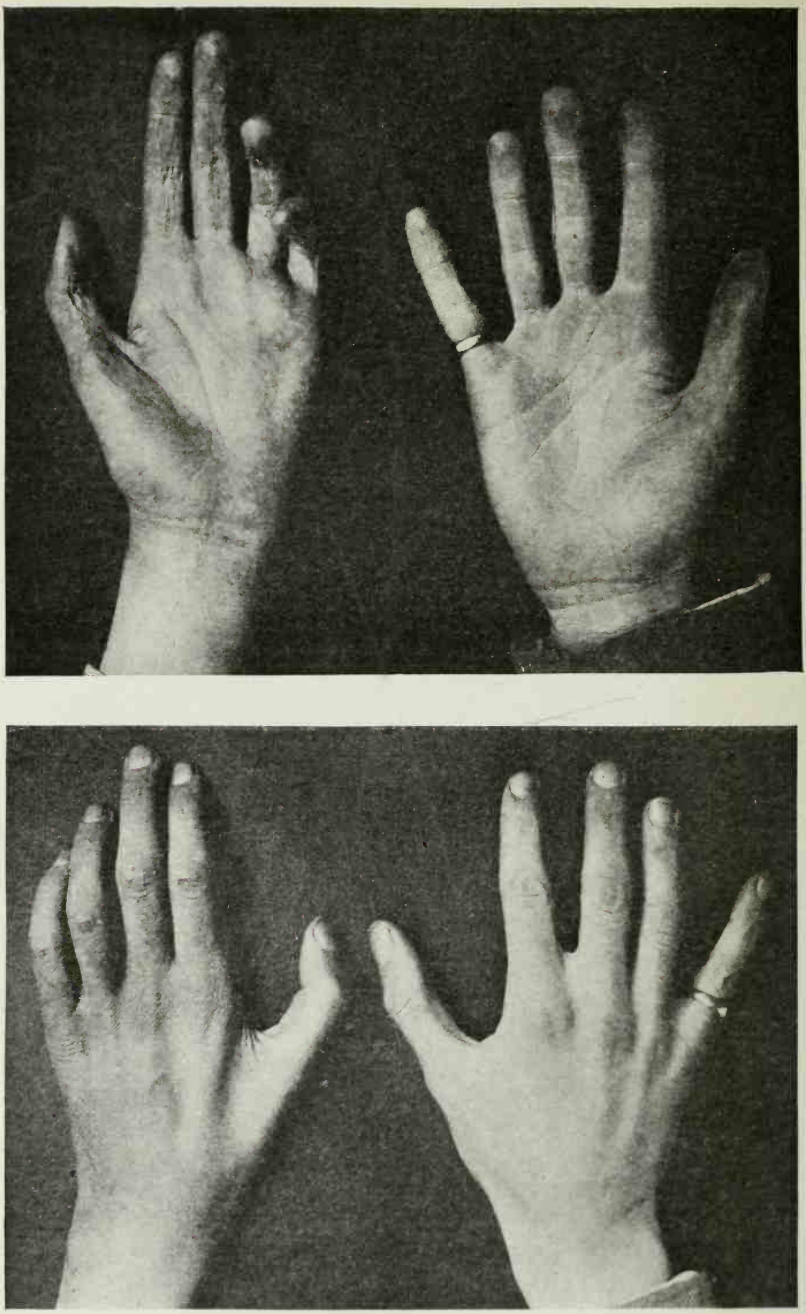


FIG. 69.—A GUNSHOT WOUND OF THE ULNAR NERVE ONE INCH ABOVE THE INTERNAL CONDYLE OF THE LEFT HUMERUS. Note the characteristic flexion of the fingers, the wasting of the interossei, of the hypothenar muscles, and of the thumb adductors. The patient is trying to abduct all his fingers; compare the two hands.

phalangeal joints of all the fingers become extended and the interphalangeal joints flexed, and this condition is more marked in the little and ring fingers, for in these we have the additional effect resulting from paralysis of the inner lumbricales; consequent too on the paralysis of the interossei, the fingers can no longer be spread out in fanlike fashion (Fig. 69); the little finger is, however, often kept widely abducted. From paralysis of the adductor obliquus and adductor transversus, the power of adducting the thumb is lost; the hypothenar muscles, the interossei, the thumb adductors, and the ulnar lumbricales waste, and the long flexor tendons become visible as longitudinal ridges in the palm. Division at or above the elbow, in addition to these symptoms, is accompanied by paralysis of the flexor carpi ulnaris and the inner half of the flexor profundus digitorum.

Vasomotor and Trophic Changes.

Horny thickening of the epidermis (hyperkeratosis) over the palmar distribution of the ulnar nerve occurs in certain long-standing cases, otherwise trophic changes are uncommon; in particular, we do not meet with the excessive sweating so characteristic of median palsy. Sometimes, when the median and ulnar are both injured, the difference as to the sweating of the two cutaneous territories is particularly striking.

Diagnosis. The symptoms of paralysis of the

ulnar nerve can only be confused with those due to a lesion of the eighth cervical and first dorsal roots, or of the inner cord of the plexus, and in these the motor and sensory paralysis have the characteristic distribution of root-areas (see p. 110); there is anæsthesia along the inner side of the forearm and hand, and all the small muscles of the hand are paralysed (Figs. 55 and 57).

MEDIAN NERVE

The median nerve arises by two heads, one from the outer cord and the other from the inner cord of the brachial plexus. The outer head receives fibres from the fifth, sixth, and seventh cervical nerves, the inner head from the eighth cervical and first dorsal nerve. The nerve descends along the outer edge of the brachial artery and crosses it in the lower half of the arm. At the bend of the elbow it lies internal to the brachial artery, and beneath the bicipital fascia and the median basilic vein. It enters the forearm between the two heads of the pronator radii teres, and runs down the forearm between the superficial and deep muscles inclining towards the radial side, and at the wrist becomes almost subcutaneous, lying directly beneath the tendon of the palmaris longus, and having the flexor carpi radialis on its radial side and the superficial flexor tendons on its ulnar side. In the forearm it supplies all the muscles with the exception of the flexor carpi ulnaris and the inner half of the

flexor profundus digitorum. It passes beneath the anterior annular ligament, lying on the radial side of the flexor tendons, and at its lower border gives off muscular branches to the abductor pollicis, opponens pollicis, and superficial head of the flexor brevis pollicis, and cutaneous branches to the palm and three and a half radial fingers.

Sensory Symptoms.

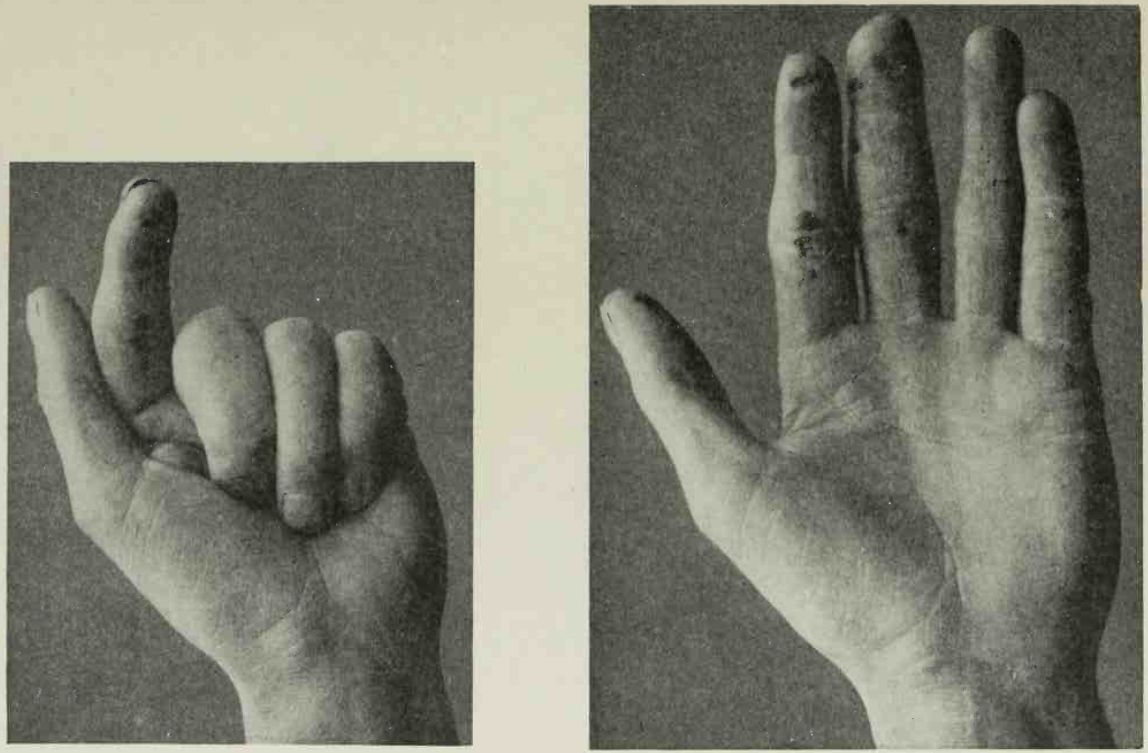
In complete median palsy cutaneous anæsthesia is present in the palmar surface of three and a half radial digits and in the corresponding part of the palm up to the fold of the wrist. The anæsthesia also extends on to the dorsum of the index, middle, and half the ring finger, except on the proximal half of the proximal phalanges which are supplied by the radial nerve (see Fig. 84, p. 165). Joint-sense is lost in the thumb, index and middle fingers, and there is generally astereognosis, i.e. inability to recognize by feeling alone the shape of objects placed between the affected digits. Total anæsthesia of the affected territory is rare; more usually we find simple blunting of sensation to touches and pin-pricks. On the other hand, this blunted cutaneous area is often hyper-sensitive to deep pressures. The extent of anæsthesia varies greatly (see Figs. 71–75).

Spontaneous pains are highly characteristic of median injuries. They tend to appear ten days or a fortnight after the injury, gradually increasing in severity week by week, until they reach a maximum. This condition may last for months; the pain then

gradually begins to subside spontaneously, and ultimately disappears. These median pains are localized chiefly in the finger-tips, at the fold of the palm with the fingers, and at the inner side of the thenar eminence. They are stabbing, crushing, and burning pains of great intensity, keeping the patient awake at nights, aggravated by heat and somewhat allayed by cold wet applications; they are intensified not merely by touching the affected hand, but also by sudden jarring of other parts of the body, by coughing or sneezing, or even by emotion of any sort; these pains are also produced by pressure over the median nerve at any part of its course below the level of the injury.

Motor Symptoms.

When the nerve is divided at or above the elbow all the muscles supplied by it are paralysed; there is paralysis and wasting of the pronators, and the forearm cannot be pronated beyond the mid-position; on flexion of the wrist the hand is drawn to the ulnar side, consequent on paralysis of the flexor carpi radialis; the long flexors of the thumb and fingers (except the ulnar half of the flexor profundus) are paralysed, and in attempting to clench the fist it will be noted that there is no flexion of the terminal phalanx of the thumb, that the index finger can only be flexed at the metacarpo-phalangeal joint, and that the ring and little fingers are the only ones whose terminal joints can be fully flexed (see Fig. 70 A).



A B

FIG. 70.—PARALYSIS OF THE LEFT MEDIAN NERVE. The nerve was incompletely divided. A shows the maximum flexion of the fingers. Note the position of the index finger, flexed only at its metacarpo-phalangeal joint; note also the inability to flex the terminal phalanx of the thumb. B shows the hand at rest; note the absence of deformity, also the multiple trophic lesions on the thumb and on the two radial fingers.

Consequent on the paralysis of the thenar muscles, the power to abduct and oppose the thumb is lost (Figs. 78 and 79).

Fig. 70 is that of a man who received a punctured wound of the left upper arm, immediately to the inner side of the biceps, and three and a half inches above the level of the internal condyle. When seen four months later there was anæsthesia to cotton-wool touches over the palmar surface of the thumb, and of the index, middle, and half the ring fingers, and over the corresponding half of the palm extending to just below the fold of the wrist. The anæsthesia to pin-prick was not quite so extensive and did not include the palmar surface of the radial half of the ring finger. On the dorsum there was anæsthesia to cotton-wool touches and pin-pricks over the terminal phalanx of the thumb, and over the terminal, middle, and half the proximal phalanges of the index, middle, and the radial half of the ring fingers. Deep pressure-sense was lost over the terminal phalanges of the index and middle fingers. Joint-sense was normal in all fingers (Fig. 71).

The patient could fully flex his three ulnar fingers, making the finger-tips touch the palm. The index finger could only be flexed at the metacarpo phalangeal joint by means of the interossei. The terminal phalanx of the thumb could not be flexed. The thumb could be opposed and abducted. There were many trophic lesions over the insensitive area. At the operation it was found that the median nerve was imbedded in a mass of fibrous

tissue ; when dissected free from this it was found that the nerve was incompletely divided, the undivided fibres forming the antero-internal segment of the nerve.

When the median nerve is divided below the elbow, i.e. below the point at which the muscles to the fore-

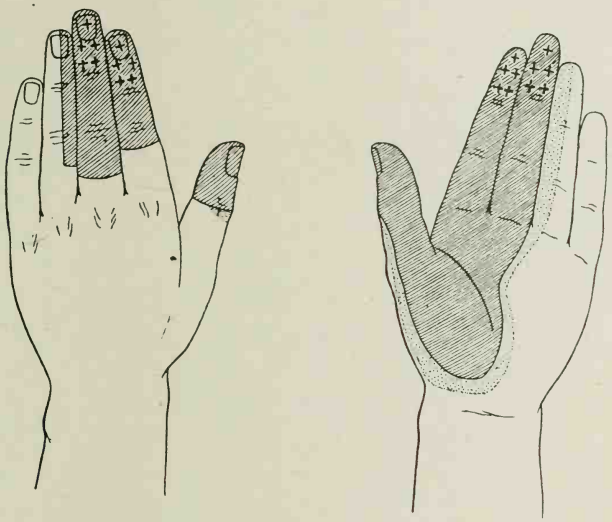


FIG. 71.—PARALYSIS OF THE MEDIAN NERVE. The dotted area is that insensitive to cotton-wool touches ; the shaded area is that insensitive to pin-pricks and cotton-wool touches. The crosses indicate the region insensitive to deep pressure.

arm are given off, then the only muscles paralysed are the abductor and opponens pollicis and the two outer lumbricales.

It is necessary to emphasize the fact that the hand when at rest shows little or no deformity, and careful routine examination is necessary lest the lesion be overlooked (see Fig. 70 B).

Paralysis of Median and Ulnar Nerves

Gunshot wounds of the arm and forearm often result in injury to both the median and the ulnar nerves (see table, p. 85).

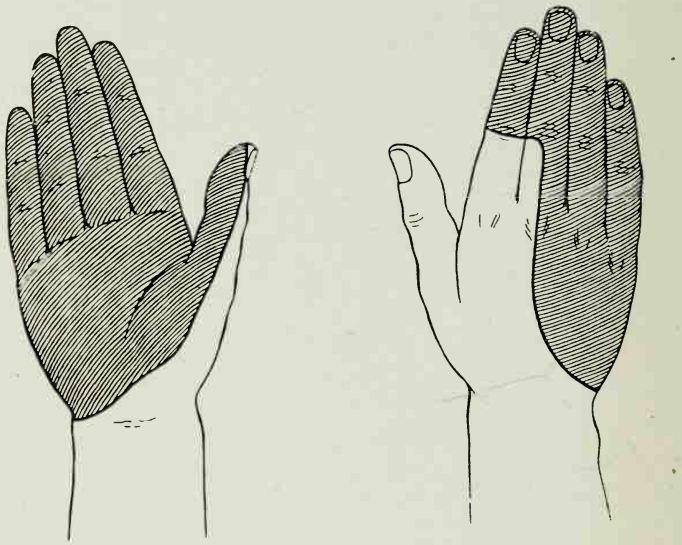


Fig. 72.—Paralysis of median and ulnar nerves. The shaded area shows the loss to pin-prick and cotton-wool. The ulnar nerve was completely divided; the median was embedded in fibrous tissue.

Sensory Symptoms.

The anæsthesia is that of the conjoint median and ulnar distribution. This varies greatly. Compare Figs. 72–75.

Fig. 72 is that of a soldier wounded in the middle of the flexor aspect of the forearm by several fragments of shell. At the operation it was found that the ulnar nerve was completely divided, and the median nerve embedded in fibrous tissue.

A soldier received a gunshot wound at Ypres in February 1915; the bullet passed through the right humerus. Entrance-wound, middle of posterior surface of arm, $4\frac{1}{2}$ inches above the olecranon; exit, inner side of arm about its middle.

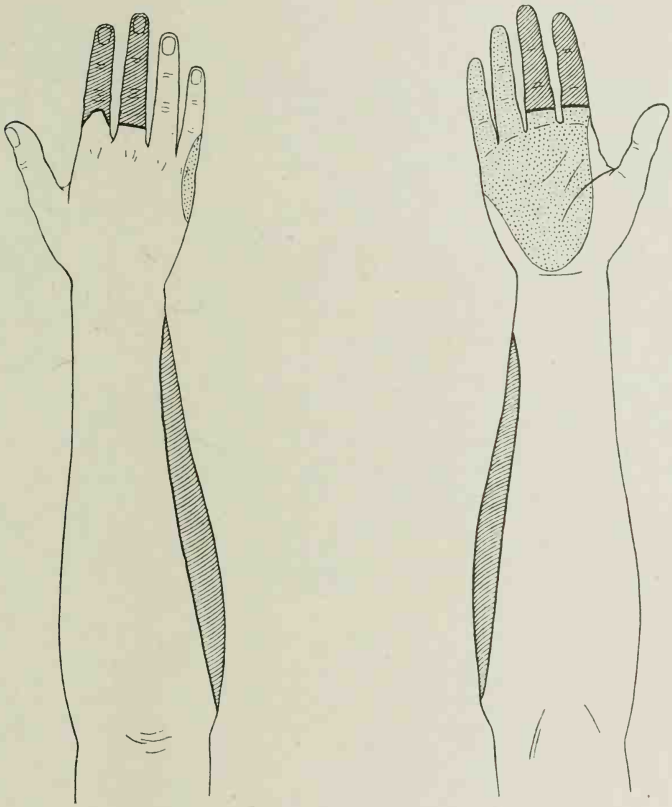


FIG. 73.—PARALYSIS OF MEDIAN, ULNAR AND INTERNAL CUTANEOUS NERVES. A soldier shot through the lower third of right humerus; at the operation the median was found completely divided, and the ulnar and internal cutaneous embedded in dense fibrous tissue. Note the small protopathic loss. The shaded area indicates protopathic and epicritic loss, the dotted area epicritic loss only.

When seen in July 1915 the exit-wound was still suppurating. The anæsthetic areas are shown in Fig. 73.

The wrist can be extended, but not flexed, supination is powerful, pronation nearly absent. The fingers can be

extended, but only partially flexed—the ring and little finger can be flexed more than the others; the thenar and hypo-

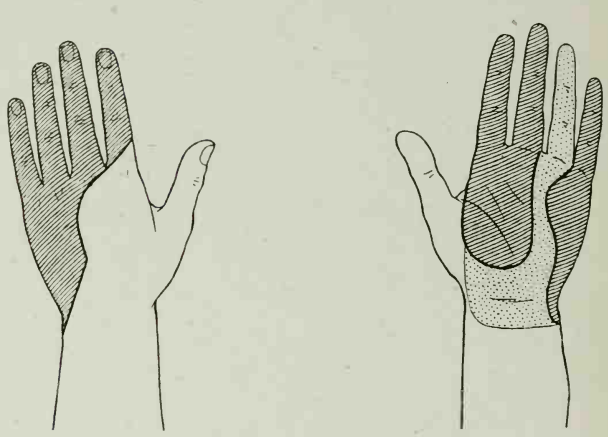


FIG. 74.—PARALYSIS OF THE MEDIAN AND ULNAR NERVES. The median was completely divided; the ulnar severely compressed.

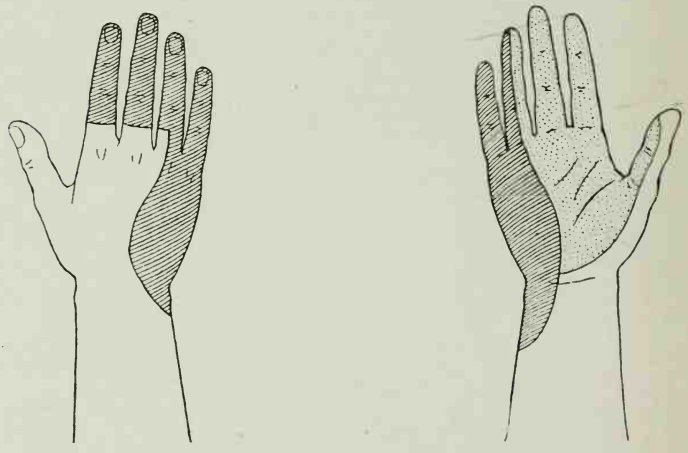


FIG. 75.—PARALYSIS OF THE MEDIAN AND ULNAR NERVES. Both nerves were compressed; their deep surfaces were continuous with a mass of scar tissue.

thenar muscles are markedly wasted; the thumb cannot be adducted, abducted, or opposed.

The anterior sinus was scraped, several pieces of bone were removed from a cavity in the humerus and the wound

sewn up. A month later the wound was opened up, and in a hole in the humerus were found the obliterated brachial artery, the two ends of the median nerve, and the ulnar and internal cutaneous nerves embedded in a dense mass of fibrous tissue. See Fig. 32.

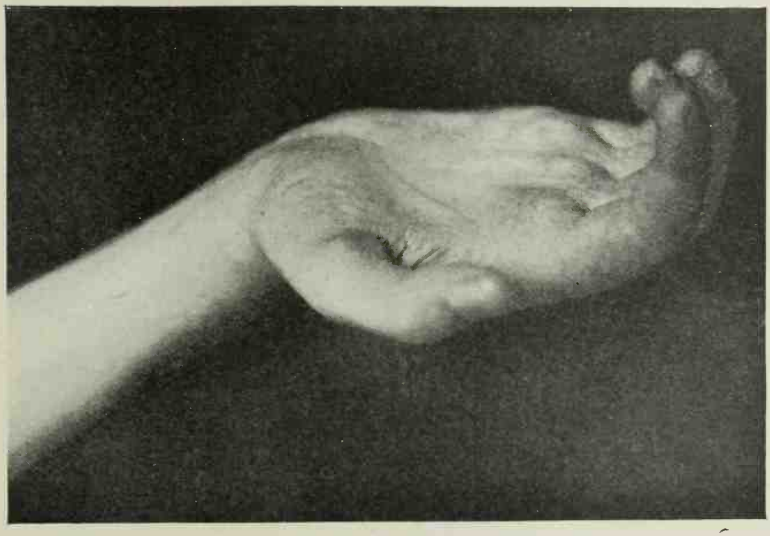


FIG. 76.—PARALYSIS OF THE MEDIAN AND ULNAR NERVES. Note the marked wasting of the palmar muscles, and the altered position of the thumb—it has fallen back into the same plane as the other fingers (*main de singe*).

Motor Symptoms.

There is paralysis of all the muscles supplied by both nerves. There is no power of flexion of the wrist or of the fingers, and the power of pronation is lost. All the thenar and hypothenar muscles are paralysed, together with the interossei and lumbricales.

The wasting of the palm is very marked (Fig. 76).

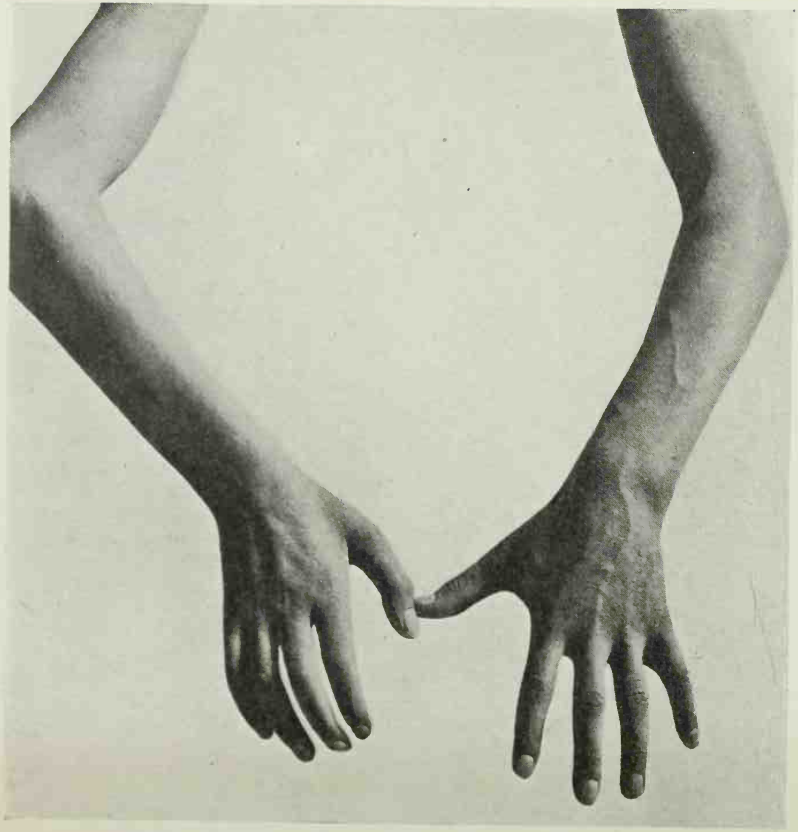


FIG. 77.—PARALYSIS OF RIGHT MEDIAN AND ULNAR NERVES, SHOWING PARALYSIS OF INTEROSSEI. The patient is strenuously attempting to abduct all his fingers.

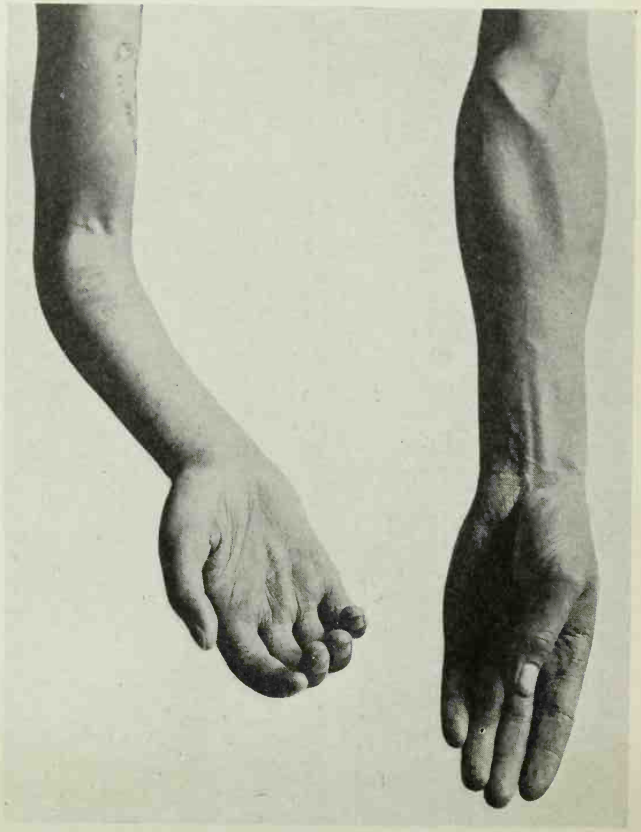


FIG. 78.—RIGHT MEDIAN AND ULNAR PARALYSIS. The patient is trying to oppose both thumbs. Note the absence of that movement in the right thumb.

FIG. 79.—RIGHT MEDIAN AND ULNAR PARALYSIS. The patient is vainly attempting to abduct his right thumb. Compare position of thumb with that of a normal abducted thumb below.

All power of abducting and adducting the fingers is lost (Fig. 77).

The patient can no longer abduct, adduct, or oppose his thumb (Figs. 78 and 79).

THE MUSCULO-SPIRAL NERVE

The musculo-spiral nerve is one of the most commonly injured nerves.

This nerve extends from the axilla, winding round the back of the humerus, to the bend of the elbow, where it divides into the radial and posterior interosseous nerves.

Whilst lying on the inner side and then on the back of the humerus the nerve gives off its muscular branches to the three heads of the triceps, and on gaining the outer side of the arm, where it lies between the brachialis anticus and supinator longus, it gives branches to these two muscles and also to the extensor carpi radialis longior.

Under cover of the supinator longus, at the bend of the elbow, it divides into the radial and posterior interosseous, through which latter nerve all the extensors of the wrist, fingers, and thumb are supplied.

The commonest site of injury is in the lower third of the arm, complicating fractures of the humerus. The nerve may also be injured in the upper part of the arm from the pressure of a crutch, or by compression of the nerve between the hanging arm and the back of a chair, or by the edge of an operating table. (See compression, p. 28.)

The posterior interosseous nerve is occasionally injured in dislocations of the head of the radius and in fractures of its upper end.

In projectile wounds the musculo-spiral nerve may be injured in any part of its course.

Motor Symptoms.

Injury to the musculo-spiral is evidenced by paralysis of the muscles supplied by it. As a rule, the nerve is injured after its branches have been supplied to the triceps muscle, so that power to extend the arm is preserved, but there is paresis of the brachialis anticus and paralysis of the supinator longus and of the extensors of the wrist, fingers, and thumb. The attitude assumed by the hand is characteristic. The hand is in a condition of 'drop-wrist'; the wrist cannot be extended, the fingers and thumb cannot be extended (Figs. 80 and 81). No possible extension of the fingers at the metacarpo-phalangeal articulations can take place; on making a forceful effort to extend the fingers, the wrist becomes flexed by the flexors of the wrist, and extension may take place at the interphalangeal joints with flexion at the metacarpo-phalangeal joints on account of the unopposed action of the interossei and lumbricales.

Fig. 80 is that of a soldier who in June 1915 received a gunshot wound of the left arm, fracturing the humerus about its middle.

When seen in July 1915 patient had a 'drop-wrist'; there was no power to extend the wrist, the fingers, or the thumb. To faradism there was no reaction in any of the

extensors, neither did they react to the strongest power of the condenser (3 microfarads).

There was no anæsthesia to pin-prick or cotton-wool. At the operation in August 1915 the musculo-spiral nerve was found completely divided; the ends were two-thirds of an inch apart, and were connected by a mass of dense fibrous tissue; there was a slight bulbous enlargement on the proximal end.

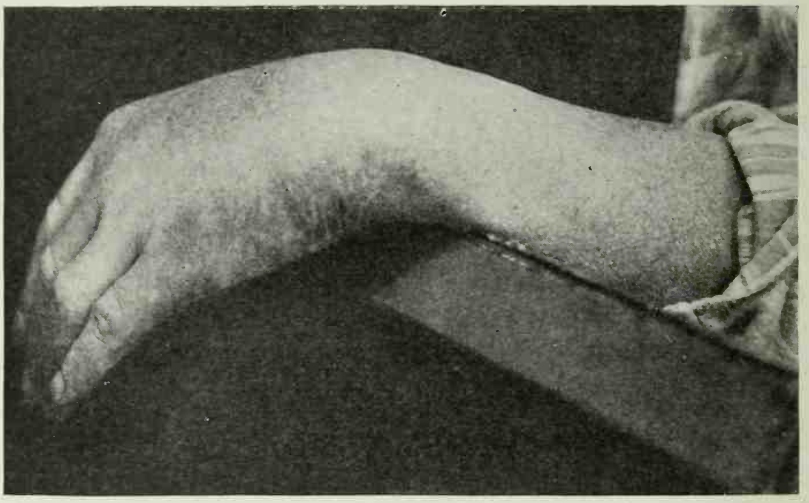


FIG. 80.—PARALYSIS OF THE MUSCULO-SPIRAL NERVE. A gunshot wound through the left upper arm, fracturing the humerus about its middle and completely dividing the nerve. The hand is in a condition of 'drop-wrist'. There is complete inability to extend the wrist, the fingers, or the thumb.

Musculo-spiral Paralysis from Compression.

Fig. 81 is that of a soldier who received a bullet wound through the triceps muscle at the level of the deltoid insertion.

The extensors of the wrist, fingers, and thumb did not react to faradism, but did to galvanism KCC > ACC, and to the condenser (3 microfarads).

At the operation the nerve was found flattened out by the fibrosed triceps; there was no solution of continuity of the nerve and no 'hardening' in the nerve itself.

Compare the electrical reactions of the preceding case, and the condition of the nerves when exposed. Three weeks after the operation the patient could feebly extend his fingers and thumb.

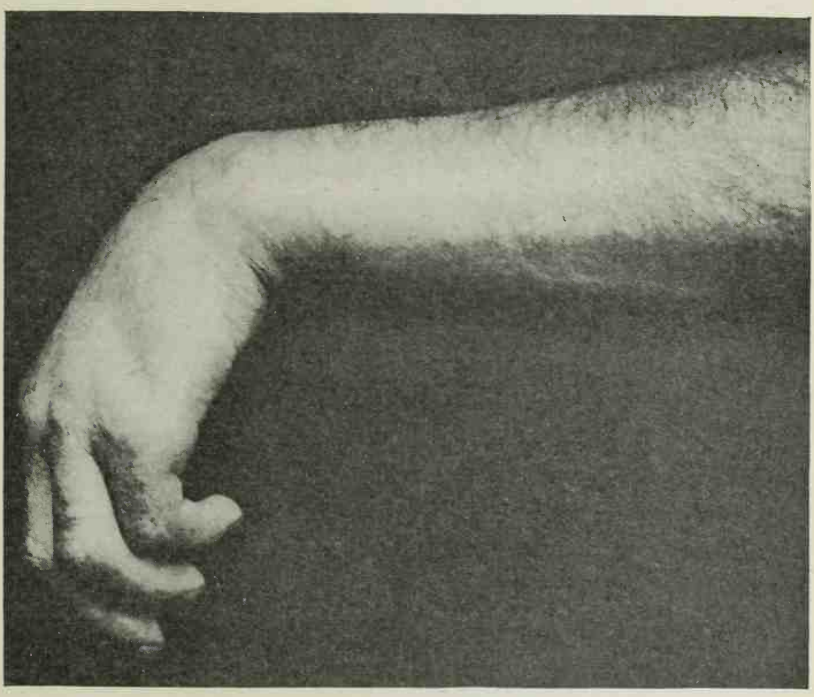


FIG. 81.—PARALYSIS OF THE MUSCULO-SPIRAL NERVE. The nerve was compressed by the fibrosed triceps muscle.

Sensory Symptoms.

Lesions of the musculo-spiral nerve in the lower third of the arm are not accompanied by anæsthesia.

A lesion in the upper part of the arm, above the origin of the external cutaneous branches, is associated with loss of sensation over the dorsum of the hand and thumb and over part of the proximal phalanges of the two and a half radial fingers (Fig. 82);

occasionally there is loss of sensation on the dorsum of the forearm on its radial side (Fig. 83).

A soldier received a bullet wound at Ypres in October. He was rising from the prone position, and the bullet entered one inch above the insertion of the deltoid. The exit was two inches lower, through the middle of the triceps. His wrist dropped at once.

Examination in December 1914. No loss of sensation to cotton-wool touches; to pin-pricks blunting on the

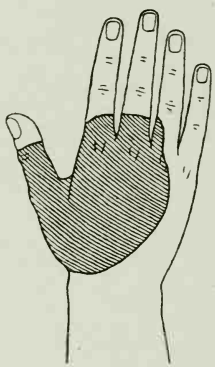


Fig. 82.—Sensory loss accompanying a lesion of the musculo-spiral nerve before its external cutaneous branches had been given off. To pin-pricks there was blunting over the shaded area. There was no epicritic loss (compare Fig. 84).

dorsum of the hand, including $3\frac{1}{2}$ radial digits as far as the middle of the proximal digits (Fig. 82). The joint-sense and vibration-sense in all the fingers normal. Movements of elbow powerful, both flexion and extension, but the supinator longus does not contract during flexion of elbow. Pronation and supination normal. Total paralysis of the wrist and fingers. Intrinsic muscles of hand normal. To faradism feeble reaction in triceps, no reaction in supinator longus or extensors of wrist and fingers. To galvanism all muscles react, KCC > ACC.

February 1915. Patient has been treated on a dorsi-flexion splint and has had regular massage and electrical

treatment. The area of loss of sensation to pin-pricks has diminished, the distal part only remaining, corresponding to the dorsum of the fingers. Supinator longus contracts feebly; extensors of wrist fair; extensors of fingers and thumb still paralysed.

April 1915. Pin-pricks as in February. Supinator longus powerful, extensors of wrist good; extensors of fingers and thumb now contract fairly well.

From the rapidity of recovery, as also from the reaction to galvanism, it is most likely that here we are dealing with a severe contusion of the musculo-spiral nerve.

In October 1914 a German soldier was wounded by a rifle-bullet. The left elbow was flexed at the moment, supporting a rifle. The bullet entered through the first interosseous space, and passed out in front close to the radial styloid process; it then re-entered the upper arm two inches above the elbow to the inner side of the biceps, and made its second exit through the posterior surface of the arm. The left humerus was fractured and badly comminuted.

At an operation for plating the humerus in December 1914 the musculo-spiral nerve was divided and immediately sutured.

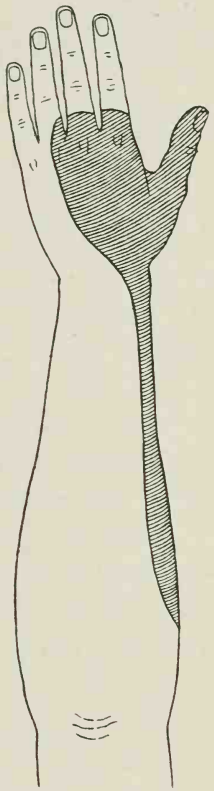


FIG. 83.—MUSCULO-SPIRAL PARALYSIS, with blunting of sensation to pin-prick and cotton-wool over the shaded area.

When seen by us in January 1915 there was blunting to cotton-wool and pin-pricks along the outer side of forearm and in the radial distribution of hand, i.e. the thumb and two and a half radial fingers (Fig. 83). There was total paralysis of the supinator longus and of the extensors of the wrist and fingers and thumb.

April 1915. Motor phenomena as before. Anæsthesia recovered except on the dorsum of the thumb and adjacent part of the hand.

POSTERIOR INTEROSSEOUS NERVE

Isolated injury of this nerve is rare.

A soldier running forward and stooping was hit by a rifle-bullet at a range of about 500 yards.

The entrance was two inches below the external condyle of the right humerus ; the exit was in the middle line of the forearm in front, two inches below the bend of the elbow. The radiogram showed severe shattering of the head and tuberosity of the radius.

When seen two months later there was no sensory loss, protopathic or epicritic, in the forearm or hand.

The joint-sense was normal ; there was doubtful diminution of vibration-sense in the right thumb only. He could flex and extend the elbow; could flex wrist, fingers, and thumb. He could not extend the wrist, fingers, nor thumb, and there was no response to faradism in the paralysed muscles.

THE RADIAL NERVE

A lesion of the radial nerve in the upper part of the forearm is not usually accompanied by anæsthesia ; if, however, it be wounded after it has been joined by branches from the external cutaneous branch of the musculo-spiral, there is anæsthesia on the back of the wrist, the proximal phalanges of the fingers, and the dorsum of the thumb. This association of nerves may take place above the middle of the forearm (Fig. 84).

Fig. 84 is that of a soldier with a rifle-bullet wound through the upper third of the left forearm. The entrance was on

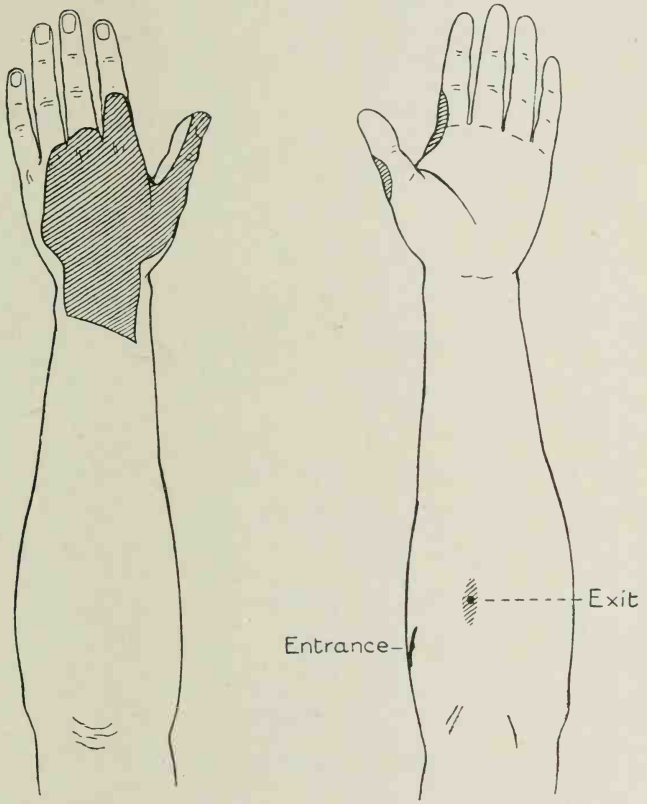


FIG. 84.—PARALYSIS OF THE RADIAL NERVE. A bullet wound of the upper third of the left forearm. The shaded area is anæsthetic to pin-prick and cotton-wool.

the radial border of the forearm, the exit in the middle line. The exit-wound was a large indurated scar closely adherent to the underlying flexor muscles. There was no motor paralysis.

CHAPTER X

THE CAUDA EQUINA

The term 'cauda equina' is given to the collection of nerve roots which occupies the spinal canal below the first lumbar vertebra, including all the lumbar, sacral, and coccygeal nerve roots.

According to the roots affected, anterior or posterior, we have motor or sensory symptoms, in each case distributed in root fashion. When the whole cauda equina is affected there is paralysis of all the muscles of the lower limbs, accompanied by anæsthesia of the whole limb extending upwards to Poupart's ligament in front, and to the upper part of the buttocks behind; the anæsthesia includes the genitals, and there is loss of control of the bladder and rectum.

As will be seen from the diagrams (Fig. 85), **the sensory areas** over the anterior, inner, and outer surfaces of the lower limb are supplied by the lumbar roots. The first lumbar root supplies the neighbourhood of Poupart's ligament and the iliac crest; the second supplies the upper part of the thigh and the buttock; the third supplies the lower part of the thigh; the fourth supplies the knee and the inner surface of the leg; and the fifth supplies the outer surface of the leg and the dorsum of the foot.

The posterior aspect of the limb is supplied by the sacral roots: the first supplies the sole of the foot and the calf; the second supplies the popliteal

surface, the back of the thigh, and the lower edge of the buttock; the third, fourth, and fifth sacral roots supply the perineum; and the coccygeal root supplies the skin over the tip of the coccyx.

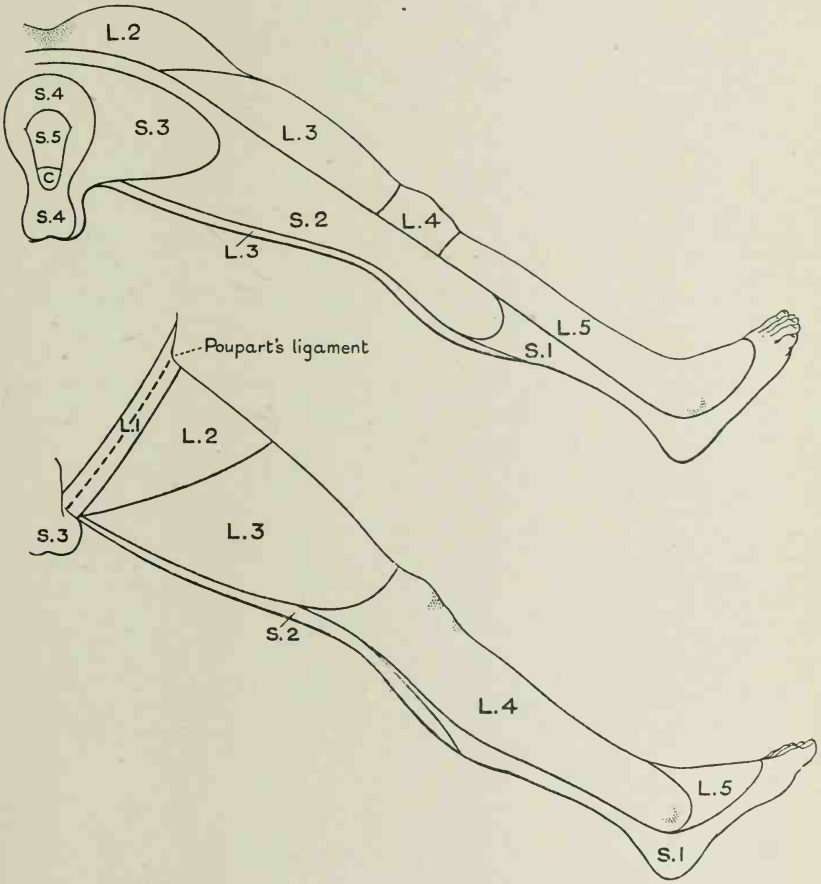


FIG. 85.—THE CUTANEOUS DISTRIBUTION OF THE CAUDA EQUINA.

Motor Distribution of the Cauda Equina.

With regard to the motor distribution of the cauda equina, it is fairly accurate to say that the muscles lying in an area of which the cutaneous supply is a

given posterior root, will receive their motor supply from a corresponding anterior root. This is shown in tabular form thus :

CAUDA EQUINA

	Sensory distribution.	*Motor distribution.*
1st and 2nd lumbar	Groin, upper part of thigh, and buttock	Psoas, iliacus, and pectineus
3rd and 4th lumbar	Anterior, inner and outer surfaces of thigh, the knee, and inner side of leg	Quadriceps extensor cruris, adductors, and tibialis anticus
5th lumbar	Antero-external surface of leg and dorsum of foot	Extensors of toes and peronei
1st sacral	Sole of foot and calf	Muscles of sole and calf.
2nd sacral	Popliteal space, posterior surface of thigh and lower edge of buttock	Hamstrings and glutei.
3rd, 4th, 5th sacral and coccygeal	Perineum and genitals	Levator ani and perineal muscles

Symptoms.

If the lesion includes the whole cauda equina there is total paralysis of the lower limbs, with total anæsthesia extending upwards to just above Poupart's ligament in front and to the upper part of the sacrum behind; there will also be anæsthesia of the genitals and loss of control of the bladder and rectum.

If the lesion be below the third lumbar root the quadriceps extensor muscles escape and the knee-jerks are preserved; the hamstrings and all the muscles below the knees are paralysed and the ankle-jerks are lost. When the lesion is below the second sacral root there is no paralysis of the lower limbs, and all the reflexes of the legs are normal, but the area of anæsthesia is very characteristic, there being

a 'saddle-shaped' area of anæsthesia on buttocks, perineum, scrotum, and penis; the anal reflexes are lost, and there is loss of control of the bladder and rectum.

A lesion below the third sacral root leaves the sphincter ani unaffected, and the only signs of nerve lesion are paralysis of the levator ani and anæsthesia of the anus and its immediate neighbourhood.

Lesions of the cauda equina are divided into upper and lower lesions, and of these the two following are examples:

Upper Cauda Lesion.

An officer taking part in an attack, consisting of spurts of running alternating with taking shelter, was lying down after one of these rushes when he was shot in the trunk; the entrance was just below and outside the angle of the right scapula, and the bullet lodged in the left hip-joint. When shot he felt as if his feet were curling up and then fell slack. At once he noticed loss of power in the right lower limb. When examined eleven days later, the right leg was still powerless; the left leg felt dead, but could be moved; there was no sphincter trouble.

Sensory loss. Total anæsthesia and analgesia of the right lower limb from the second lumbar to the first sacral area inclusive; all forms of cutaneous sensation being lost—touch, pain, and temperature (Fig. 86). In the left lower limb there is moderate diminution of all forms of sensation in the distribution of the fifth lumbar and first sacral areas. Joint-sense is lost in all the joints of the right lower limb from the hip downwards; in the left limb joint-sense is lost in the toes; elsewhere it is normal.

Motor loss. Total flaccid paralysis of right lower limb at all the joints. Left lower limb feeble at all joints, but no individual movement impossible.

Knee-jerks: left present, right absent.

Ankle-jerks: both absent. Plantar reflexes absent. Sphincters normal.

Electrical reaction: to faradism the right calf muscles react normally; no reaction in any other muscles of the

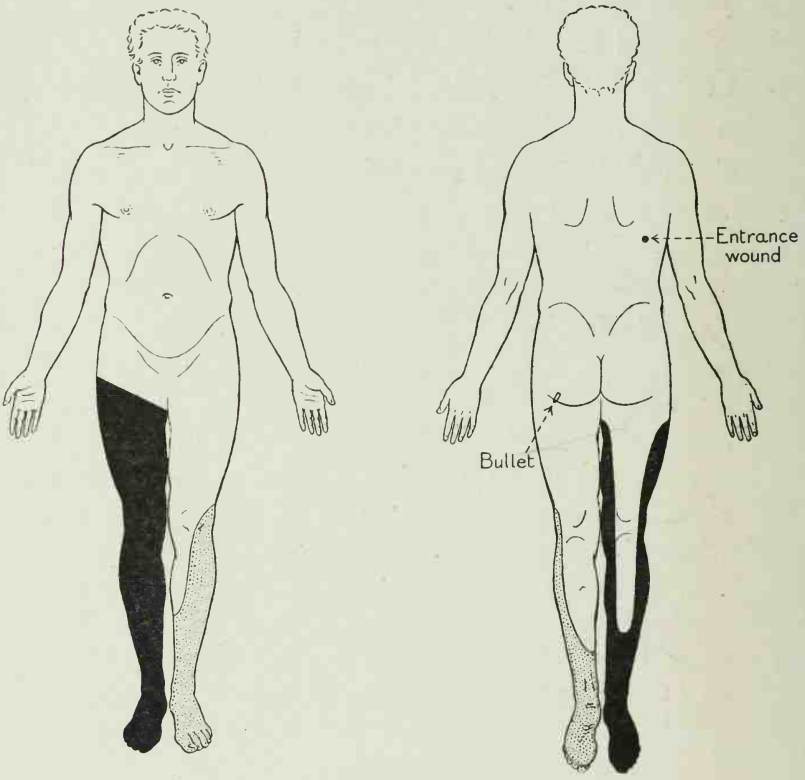


FIG. 86.—UPPER CAUDA LESION. In the right limb all forms of cutaneous sensation were lost, from the second lumbar to the first sacral area inclusive. In the left limb there was moderate diminution of these in the fifth lumbar and first sacral areas.

thigh or leg. All muscles of the left lower limb react normally.

One year later there was some return of power in all the muscles, and the only anæsthesia persisting was a small patch on the front of the right thigh.

Lower Cauda Lesion.

Two years previously a labourer when at work was injured by a fall of earth which struck his back in the lumbar region. He was buried up to his waist, and had to be drawn out. He

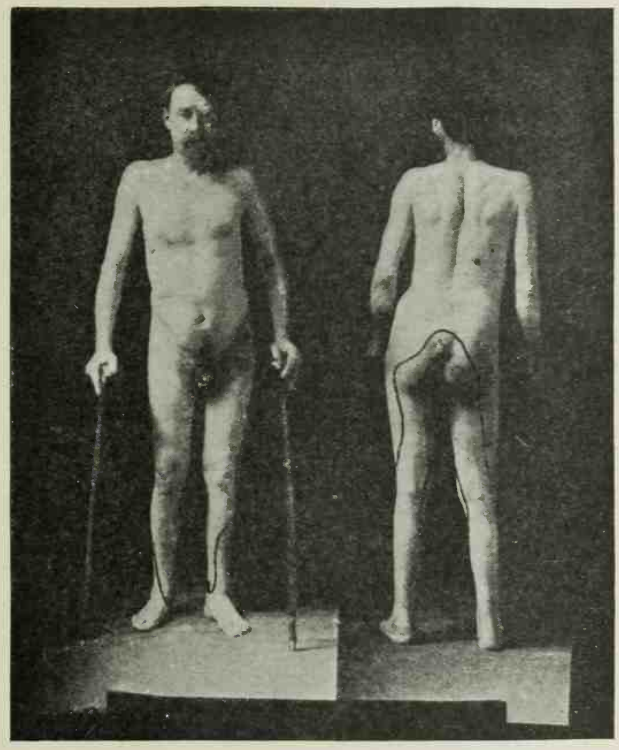


FIG. 87.—LOWER CAUDA LESION. Caused by a fall of earth which fractured the fifth lumbar vertebra and the sacrum. The area outlined is anæsthetic to pin-prick and cotton-wool, and includes the fifth lumbar root and all the roots below it.

states that his legs were limp and powerless, and that he had loss of feeling up to the waist.

During these two years a catheter was passed twice daily. Now, catheterization is not necessary, and the patient has complete control over micturition. There is still occasional incontinence of fæces. Patient has regained

considerable power in his right lower limb and can now stand. The left leg is, however, still weak. A radiogram taken three months ago showed a fracture of the fifth lumbar vertebra and of the sacrum. There is anæsthesia and analgesia from the fifth lumbar root downwards on both sides (Fig. 87), slightly lower on the right side; the epicritic loss is more extensive than the protopathic. The genitals are analgesic, but not anæsthetic.

Motor functions: the patient can feebly move the toes and ankles of both feet, the left feebler than the right. All other movements of the lower limbs are good. There is a tendency to left-sided drop-foot. Knee-jerks: right brisk, left absent. Ankle-jerks absent. Plantar reflexes absent. Cremaster reflexes present. There is an unhealed sacral bed-sore.

Lower Cauda and Conus Medullaris Lesion.

A Belgian officer was shot in October 1914. The bullet entered from the right side, one inch above the right iliac crest in the mid-axillary line, and came out at a corresponding point on the opposite side, just hitting the crest of the ilium. The patient was placed on a horse and managed to ride away, being able to move his hips and knees, but feeling marked weakness of the ankles, especially on the right side. Ever since the injury he has had anæsthetic incontinence of both sphincters. Sensory functions (Fig. 88): total loss to touch, pain, and temperature on left side of sacrum, back of left thigh, and left side of genitals, i.e. in the distribution of the second sacral root area and all the roots below; on the right side there is partial blunting of all forms of sensation in the distribution of the third sacral root and downwards, including right buttock, right saddle area, and right side of genitals.

No paralysis of individual movements of lower limbs, although all are slightly feeble; those on the right are stronger than those on the left side.

Knee-jerks present, ankle-jerks absent; bulbo-cavernosus and anal reflexes are absent.

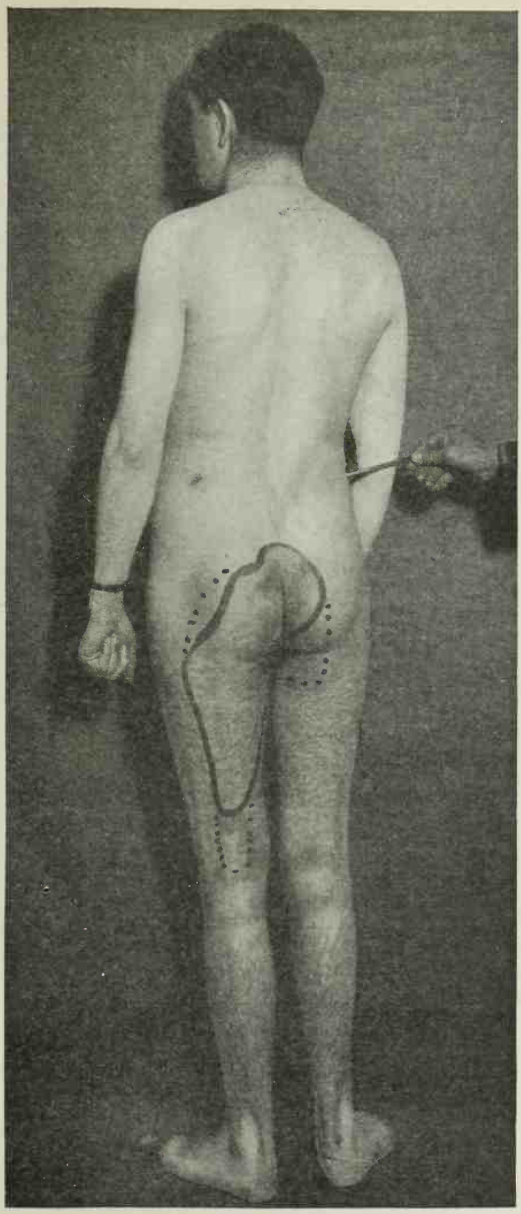


FIG. 88. LESION OF LOWER CAUDA AND CONUS MEDULLARIS. The pencil rests on the entrance-wound; the exit-wound is obvious. The anæsthesia on the left side is that of total loss to touch, pain, and temperature, and corresponds to the second sacral root and all the roots below it; on the right side there is partial blunting to all forms of sensation, and corresponds to the third sacral roots and all the roots below it. Six months previously the anæsthesia extended to the dotted line.

To faradism the sphincter ani does not contract.

November 1914. Has considerably recovered sensation on the right side of genitals. Developed two bed-sores, one on each side of sacrum.

December 1914. Walks feebly with support.

February 1915. Walks fairly well without a stick.

June 1915. Can walk well on tip-toes. Can pass water by straining, without a catheter. There is anæsthesia of the urethra with occasional dribbling of urine. There is no control over the bowel. The bed-sores are healed.

With regard to these cases it is worth noting that all three have slowly but steadily improved without operative interference.

In making a diagnosis of lesions in this neighbourhood the chief difficulty is to distinguish between lesions of the cauda equina and those of the conus medullaris (i. e. that part of the spinal cord which extends below the third sacral segment and which is situated opposite the first lumbar vertebra).

Paralysis of the bladder and rectum, combined with an area of anæsthesia corresponding to the fourth and fifth sacral and first coccygeal roots, are characteristic of a conus lesion. In both lesions there is loss of sexual power, and of bladder and rectum control.

Non-traumatic cauda lesions are more often gradual in onset than conus affections, and are usually accompanied by intense sacral root pains, and the ultimate anæsthesia of a progressive cauda affection is frequently preceded by cutaneous hyperæsthesia. A bed-sore is commoner in a medullary lesion than in a cauda lesion.

Any dissociation of anæsthesia, such as analgesia or thermo-anæsthesia without tactile anæsthesia, points to a medullary lesion. If both cauda and conus are affected, the cauda symptoms mask the others. If a lesion be asymmetrical, it is more likely to be of the cauda than of the conus.

THE GREAT SCIATIC NERVE

The sacral plexus is formed by the anterior divisions of the fourth and fifth lumbar nerves and the first and parts of the second and third sacral nerves. These nerves lie on the back wall of the pelvis upon the pyriformis muscle; they converge and form a broad band which passes into the buttock through the great sacro-sciatic foramen as the great sciatic nerve.

The great sciatic nerve is really two nerves, the internal popliteal (tibial) nerve and the external popliteal (peroneal) nerve. These are loosely held together for a greater or lesser distance by a common investing sheath; on opening this the two nerves can be traced up to the plexus, from which they take their origin by distinct and separate roots. Frequently there is no common investing sheath, and the two nerves are distinct right up to their origin; sometimes, indeed, the nerves are separated by fibres of the pyriformis muscle. It is well, then, to look upon the sciatic as two separate nerves, for not only is this true anatomically, but clinically it is very obvious.

When trauma is applied to the great sciatic nerve the resulting lesion may be of the whole nerve or of its peroneal or tibial component; in the latter case it is the peroneal nerve that almost invariably suffers. Thus, in our 48 cases of injury to the sciatic nerve,

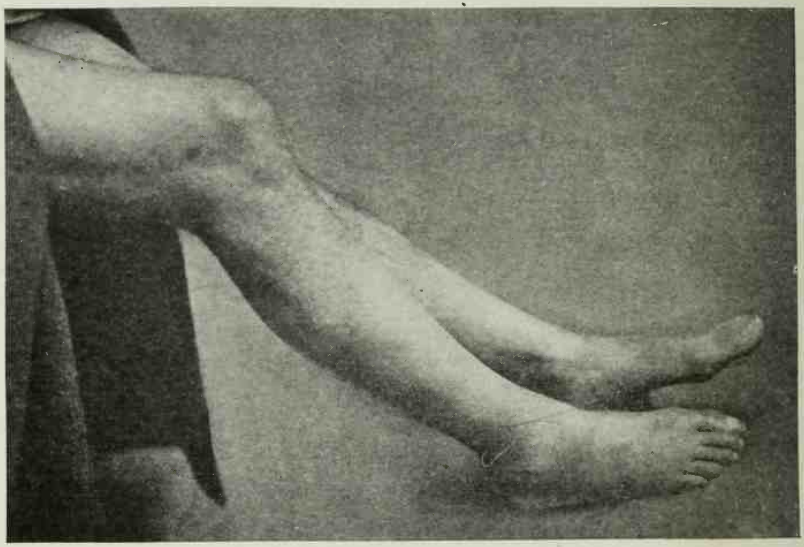


FIG. 89.—PARALYSIS OF THE RIGHT GREAT SCIATIC NERVE, FOLLOWING A GUNSHOT WOUND WHICH RESULTED IN ITS COMPLETE DIVISION. There was paralysis of all the muscles below the knee. For anæsthesia, see Fig. 90. Note the small amount of flexion of the knee-joint necessary for direct suture of the divided ends—although these, after freshening, had been 7 centimetres apart (see Fig. 19).

the whole trunk was affected in 21, the peroneal nerve in 26, and the tibial nerve in one case only. Makins pointed out that in lesions of the great sciatic nerve occurring in the South African War the peroneal fibres suffered in 90 per cent. of the cases. In this connexion Sherren points out that the peroneal fibres suffer more than the tibial

fibres in conditions such as infantile paralysis and toxic neuritis.

We do not know the reason for this, but it is an established fact that the peroneal nerve is more vulnerable than the tibial nerve.

Paralysis of the Great Sciatic Nerve.

In a case of complete sciatic paralysis there is anæsthesia of the outer surface of the leg and of the dorsum and sole of the foot; in fact, the only part of the leg which is not anæsthetic is the inner surface of the leg and ankle, which region is supplied by the internal saphenous nerve. All the muscles below the knee are paralysed; no movement of the ankle is possible—either of flexion, extension, inversion, or eversion, nor is there any movement possible in the toes, whether of flexion or extension.

If the lesion be high up the nerve trunk, the hamstring muscles are paralysed, but some power of flexion of the knee is retained owing to the gracilis being intact, receiving as it does its supply from the obturator nerve.

Fig. 89 is that of a soldier wounded near Neuve Chapelle in May 1915. The entrance-wound is on the outer side of the thigh, three inches above the head of the fibula; the exit is on the posterior surface of the thigh, four and a half inches above the popliteal crease and to the inner side of the mid-line.

At the operation in July 1915 the right sciatic nerve was found completely divided, see Fig. 19, p. 69.

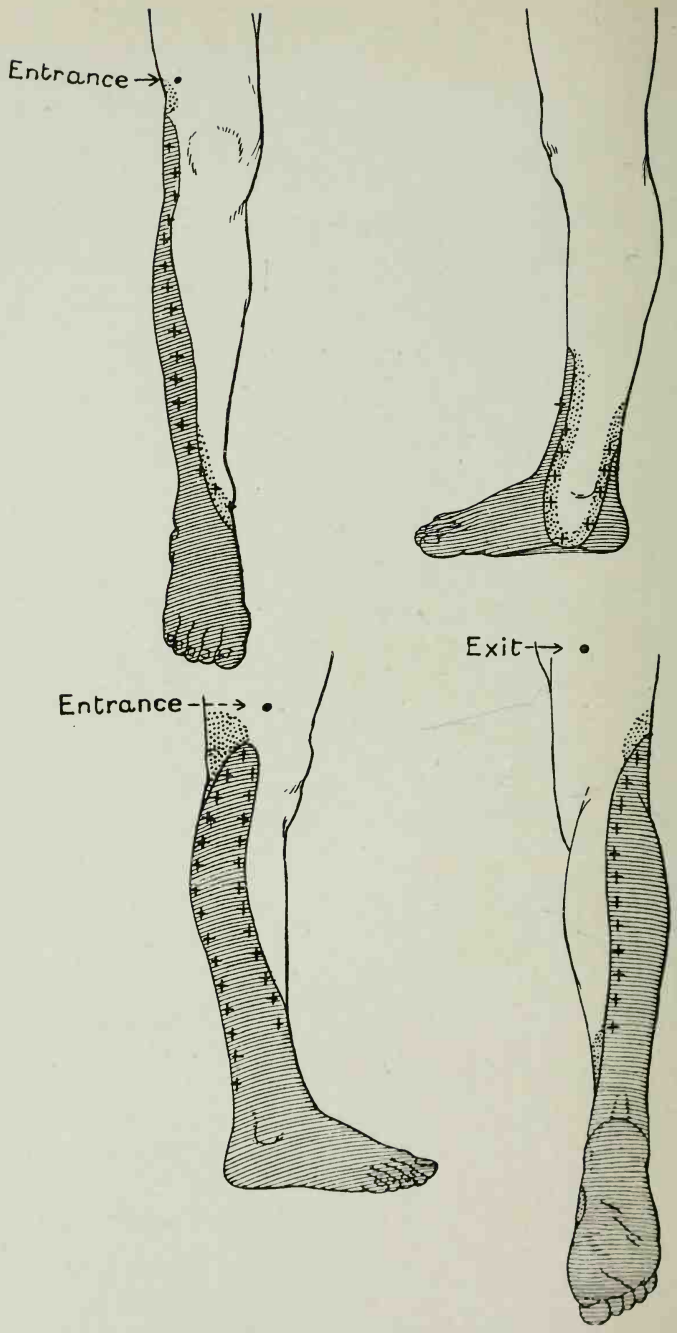


FIG. 90.—THE SENSORY LOSS IN PARALYSIS OF THE GREAT SCIATIC NERVE. The shaded area is that insensitive to pin-prick, the dotted area is the additional area insensitive to cotton-wool; the crosses mark the loss of sensation to deep pressure.

THE EXTERNAL POPLITEAL NERVE

Injury to this nerve is very common. It results from cutting accidents, fractures of the femur, fractures of the upper end of the fibula, and, most commonly, from gunshot wounds. It may be injured in any part of the course of the great sciatic nerve from the sciatic notch downwards, or in the popliteal space under cover of the biceps tendon, or as it winds round the neck of the fibula to pass from the popliteal space to the front of the leg; in this latter position it may be injured by the pressure of a Clover's crutch. It has been cut when the biceps has been tenotomized, and removal of the upper portion of the fibula has resulted in damage to the nerve (see Fig. 93).

Motor symptoms. Owing to paralysis of the peronei, tibialis anticus, and extensors of the toes, the foot cannot be dorsiflexed nor everted, the toes cannot be extended, and the foot assumes the position of talipes equino-varus; it is said to be in a state of 'foot-drop' (Fig. 91).

Sensory phenomena. There is loss of sensation over the areas supplied by the musculo-cutaneous nerve and the lateral cutaneous branch of the external popliteal nerve, i. e. over the outer side of the leg and the dorsum of the foot (Fig. 92).

Fig. 92 shows the area of anæsthesia in a soldier wounded in May 1915, at Festhubert, by a piece of shell. The entrance-wound is in the right buttock on a level with the

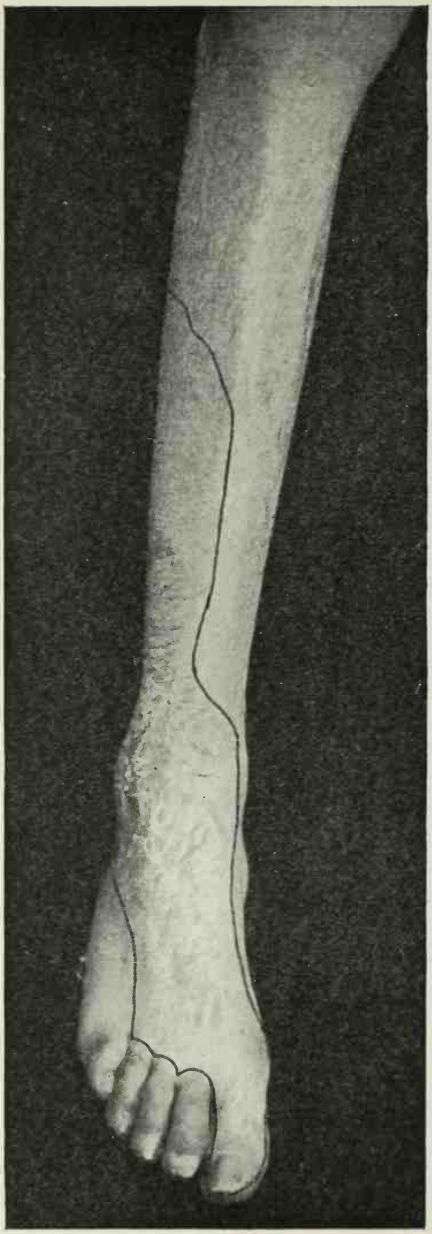


FIG. 91.—EXTERNAL POPLITEAL PARALYSIS, FOLLOWING A GUNSHOT WOUND OF THE RIGHT BUTTOCK WHICH DIVIDED THE PERONEAL HALF OF THE GREAT SCIATIC NERVE. The outlined area is that anæsthetic to pin-prick and cotton-wool. There is paralysis of the peronei, tibialis anticus, and extensors of the toes. Note the wasted condition of the extensors and peronei, the condition of 'drop-foot', and the delayed desquamation over the anæsthetic area.

top of the great trochanter and $2\frac{1}{2}$ inches from the mid-vertebral line: there is no exit-wound, but in the radiogram a piece of shell can be seen lying near the neck of the right femur.

At the operation in July 1915 it was found that the external popliteal nerve had been shot through about

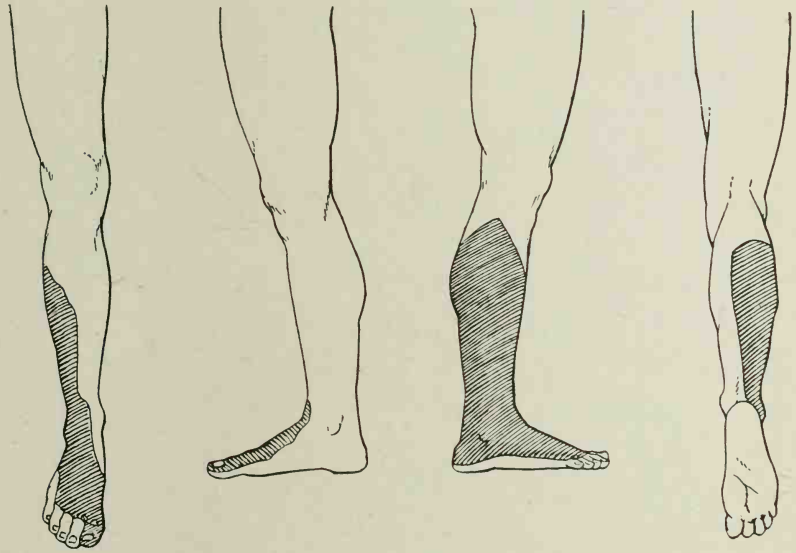


FIG. 92.—EXTERNAL POPLITEAL PARALYSIS. The shaded area shows the extent of anæsthesia to pin-prick and cotton-wool. The nerve was injured by a piece of shell one inch below the sciatic notch. There was paralysis of the peronei, tibialis anticus, and extensors of the toes, with inability to dorsiflex the foot, evert the foot, or to extend the toes.

one inch below the sciatic notch. In this situation it was replaced by a mass of fibrous tissue which was adherent to the internal popliteal nerve. This area was excised and the ends of the nerve sutured.

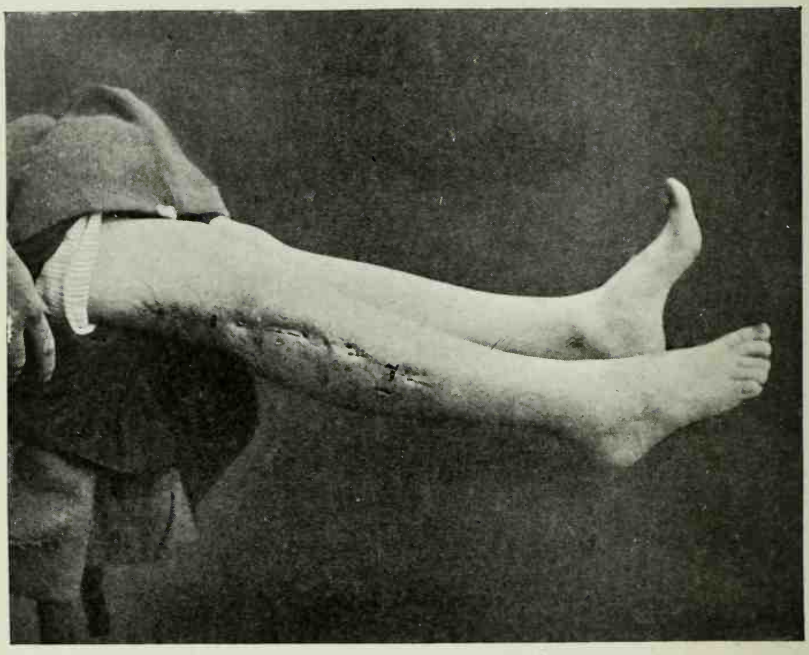


FIG. 93.—PARALYSIS OF THE RIGHT ANTERIOR TIBIAL NERVE. This nerve was injured during the removal of a large myeloid sarcoma of the upper end of the fibula. The tibialis anticus, extensor communis digitorum, and the extensor longus hallucis are paralysed. The patient is attempting to dorsiflex both feet. The right foot is in the condition of 'foot-drop'. There is no sensory loss.

THE ANTERIOR TIBIAL NERVE

Paralysis of the anterior tibial nerve is very seldom met with in civil practice, but may occur from gunshot wounds of the interosseous space.

The symptoms are those of external popliteal paralysis, with this exception—that there is no sensory loss.

Fig. 93 is that of a female patient, the upper end of whose fibula was removed for a huge myeloid sarcoma. The first stage of the operation consisted in thoroughly exposing the external popliteal, the musculo-cutaneous, and the anterior tibial nerves; these were dissected out and displaced forwards. It was thought that these had been carefully protected from injury during the whole operation, which was a very tedious and prolonged one; but much manipulation of the interosseous space was necessitated by the ramification of the growth and by the free bleeding which followed the removal of the tourniquet, and the anterior tibial nerve must have been injured, for subsequently it was found that the patient had 'foot-drop'.

Motor symptoms. There was no voluntary movement in the tibialis anticus, the extensor communis digitorum, nor in the extensor longus hallucis.

These muscles gave no response to faradic shocks, and there was reaction of degeneration to the galvanic current.

Sensory phenomena. There was no sensory loss in foot or leg, not even in the first dorsal interosseous space.

THE INTERNAL POPLITEAL NERVE

Isolated injury of the internal popliteal nerve is very uncommon. In the thigh, whilst forming part of the great sciatic nerve, it supplies muscular branches

to the quadratus femoris, gemelli, obturator internus, and the hamstring muscles. In the popliteal space it gives off branches to the gastrocnemius, plantaris, soleus, popliteus and tibialis posticus muscles.

Whilst passing down the back of the leg as the posterior tibial nerve, muscular branches are given to the soleus, tibialis posticus, flexor longus digitorum and flexor longus hallucis muscles. Beneath the internal annular ligament the posterior tibial nerve divides into the internal and external plantar nerves, and through these branches it supplies all the muscles of the sole of the foot, and sensation over the heel, sole of foot, and plantar surfaces of the toes (Fig. 94).

Injury of the nerve results in paralysis of the muscles of the calf and of the sole of the foot, and the following movements are impossible: plantar flexion of the foot, inversion of the foot, and flexion of the toes.

Posterior Tibial Nerve.

A soldier in August 1915, at Suvla Bay, was wounded by a rifle bullet in the right leg. The entrance was in front, five and a half inches above the inter-malleolar line. The exit was on the inner side, three and a half inches above the tip of the internal malleolus, and on a line with its posterior edge. The bullet had fractured the tibia. There was paralysis of the muscles of the sole of the foot,

and loss of sensation, both protopathic and epicritic, over the heel, sole, and plantar surfaces of the toes. There was loss of sensation to deep pressure over most of the heel (Fig. 94). At the operation it was found that the nerve was completely divided; there was a bulb at each end; the ends were

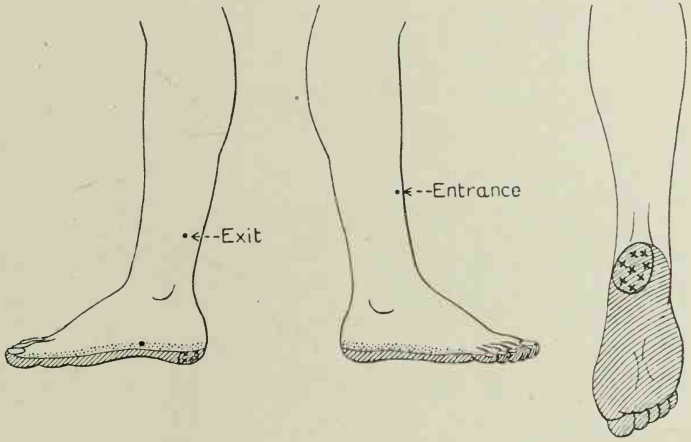


FIG. 94.—PARALYSIS OF THE POSTERIOR TIBIAL NERVE. There was paralysis of the muscles of the sole. The shaded area denotes loss of epicritic and protopathic sensation; the dotted area indicates loss of epicritic sensation alone. The crosses indicate the area over which deep sensation was lost.

separated for two and a half centimetres, and were embedded in a mass of dense fibrous tissue which was intimately adherent to the site of the fracture and also to the scar of exit. After freshening the divided ends of the nerve, they were 'bridged' by many strands of catgut, and wrapped in cargile membrane.

THE SMALL SCIATIC NERVE

Lesions of the small sciatic nerve are evidenced by anæsthesia of the lower portion of the buttock and of the posterior surface of the thigh—sometimes, too, of the popliteal area.

A soldier received a bullet wound in the buttock at Ypres in April 1915.

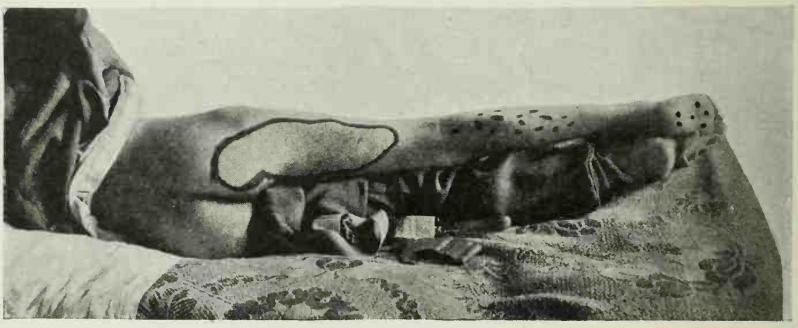


FIG. 95.—PARALYSIS OF THE SMALL SCIATIC NERVE AND PAINFUL INTERSTITIAL NEURITIS OF THE GREAT SCIATIC (internal popliteal nerve), consequent on a shell wound of the right buttock, which resulted in adhesions of the sheath of the great sciatic nerve on the inner side, and compression of the small sciatic nerve in a mass of fibrous tissue. There is anæsthesia of the lower portion of the buttock and posterior surface of the thigh; also marked cutaneous hyperæsthesia in the calf and sole of the foot (the dotted areas), and wasting of the muscles in these regions.

There was anæsthesia over the buttock and posterior surface of the thigh, and general wasting of the glutei, hamstrings, and calf muscles; there was tenderness on pressure in the calf and marked hyperæsthesia in the right sole. At the instant he was wounded he thought some one had dropped a box of ammunition on his foot, and felt sure his heel had been crushed. The pain in his foot was so

great that his boot had to be cut away; he could not bear to have it pulled off. The pain increased for two weeks, and remained at its maximum for three weeks. When seen in July the sole of the foot was so exquisitely tender that he could only rest it in bed when lying on its side; he could allow nothing to touch the sole.

There was paralysis of the small sciatic nerve, and painful interstitial neuritis of the tibial nerve.

At the operation in July 1915 it was found that immediately outside the sciatic notch the great sciatic nerve had been injured on its inner edge; here the sheath was adherent to a mass of scar tissue, in which was compressed the small sciatic nerve.

THE ANTERIOR CRURAL NERVE

Injury of the anterior crural is rare. It can be caused by bayonet, sword, shell, shrapnel, and rifle-bullet wounds. The motor loss after division is very definite, namely, inability to extend the knee, consequent on paralysis of the quadriceps extensor cruris. The pectineus and sartorius muscles are also paralysed.

From affection of the middle and internal cutaneous and the long saphenous nerves, there is anæsthesia of the front of the thigh and knee, and of the inner side of the leg extending to about $1\frac{1}{2}$ inch below the internal malleolus (Figs. 96 and 97).

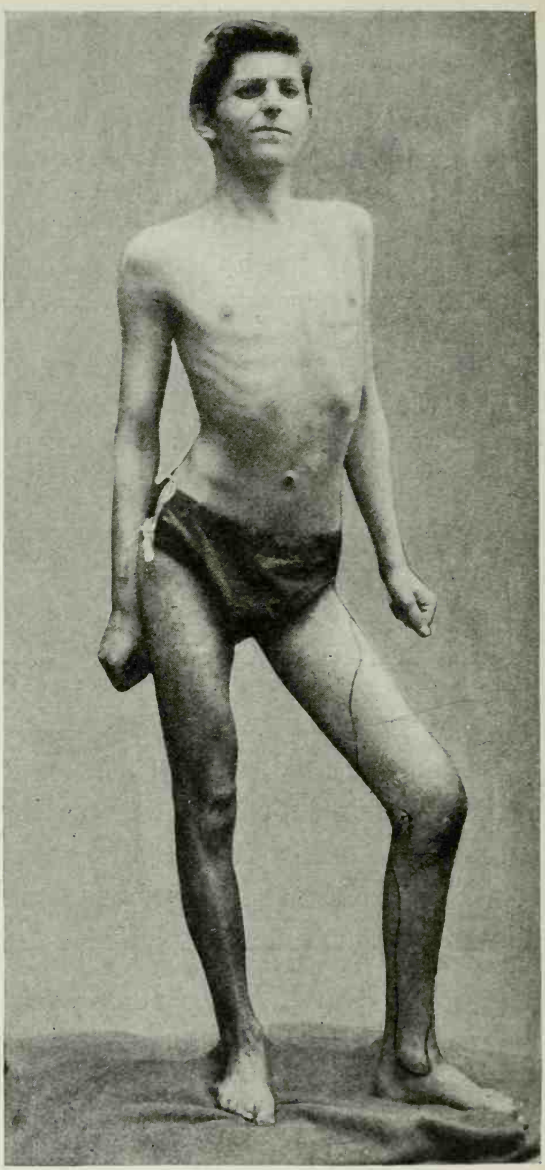


FIG. 96.—PARALYSIS OF THE LEFT ANTERIOR CRURAL NERVE, SHOWING ATROPHY AND PARALYSIS OF THE QUADRICEPS EXTENSOR CRURIS. The black line indicates the limit of anæsthesia, both epicritic and protopathic. The condition was due to hæmorrhage into and about the anterior crural nerve, in a case of hæmophilia. The left knee joint is distended with blood.

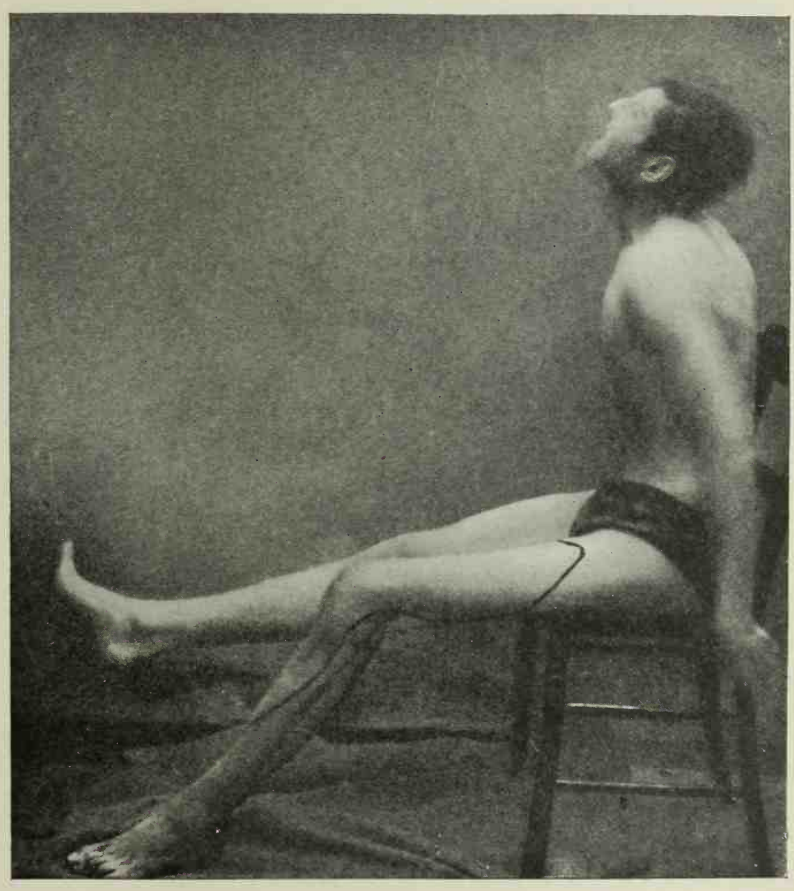


FIG. 97.—PARALYSIS OF THE LEFT ANTERIOR CRURAL NERVE. The patient is trying to extend both knees, but fails with the left.

THE OBTURATOR NERVE

Injury of the obturator nerve is rare. The diagnosis of its complete division is not difficult, seeing that all the adductors are supplied by it. The lesion is not accompanied by any area of anæsthesia.

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